SERUM MAGNESIUM LEVELS IN ACUTE MYOCARDIAL INFARCTION IN RELATION TO ARRHYTHMIAS IN PATIENTS ADMITTED IN GOVT. KILPAUK MEDICAL COLLEGE & HOSPITAL

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In partial fulfillment of regulations for award of the degree of

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BONAFIDE CERTIFICATE

This is to certify that dissertation named **"SERUM MAGNESIUM LEVELS IN ACUTE MYOCARDIAL INFARCTION IN RELATION TO ARRHYTHMIAS IN PATIENTS ADMITTED IN GOVT. KILPAUK MEDICAL COLLEGE & HOSPITAL"** is a bonafide work performed by Dr. Prabha G., post graduate student, Department of Internal Medicine, Kilpauk Medical College, Chennai-10, under my guidance and supervision in fulfillment of regulations of the Tamilnadu Dr. M.G.R Medical University for the award of M.D. Degree Branch I (General Medicine) during the academic period from May 2012 to April 2015.

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DECLARATION

I solemnly declare that this dissertation **"SERUM MAGNESIUM LEVELS IN ACUTE MYOCARDIAL INFARCTION IN RELATION TO ARRHYTHMIAS IN PATIENTS ADMITTED IN GOVT. KILPAUK MEDICAL COLLEGE & HOSPITAL"** was prepared by me at Government Kilpauk Medical College and Hospital, Chennai, under the guidance and supervision of Prof.Dr.R.Sabarathanavel, M.D., Professor and HOD, Department of Internal Medicine, Kilpauk Medical College, Chennai.

This is dissertation is submitted to The Tamil Nadu Dr.M.G.R. Medical University, Chennai in partial fulfillment of the University regulations for the award of the degree of M.D. Branch-I (General Medicine).

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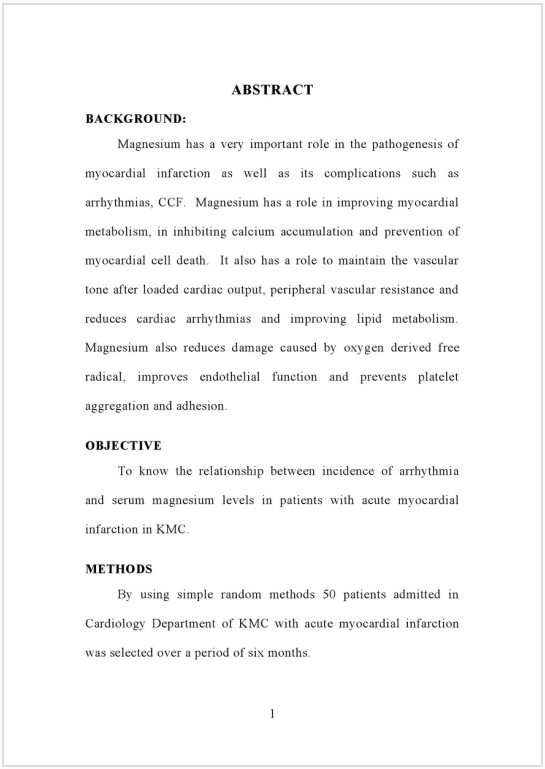

ABSTRACT

BACKGROUND:

Magnesium has a very important role in the pathogenesis of myocardial infarction as well as its complications such as arrhythmias, CCF. Magnesium has a role in improving myocardial metabolism, in inhibiting calcium accumulation and prevention of myocardial cell death. It also has a role to maintain the vascular tone after loaded cardiac output, peripheral vascular resistance and reduces cardiac arrhythmias and improving lipid metabolism. Magnesium also reduces damage caused by oxygen derived free radical, improves endothelial function and prevents platelet aggregation and adhesion.

OBJECTIVE

To know the relationship between incidence of arrhythmia and serum magnesium levels in patients with acute myocardial infarction in KMC.

METHODS

By using simple random methods 50 patients admitted in Cardiology Department of KMC with acute myocardial infarction was selected over a period of six months.

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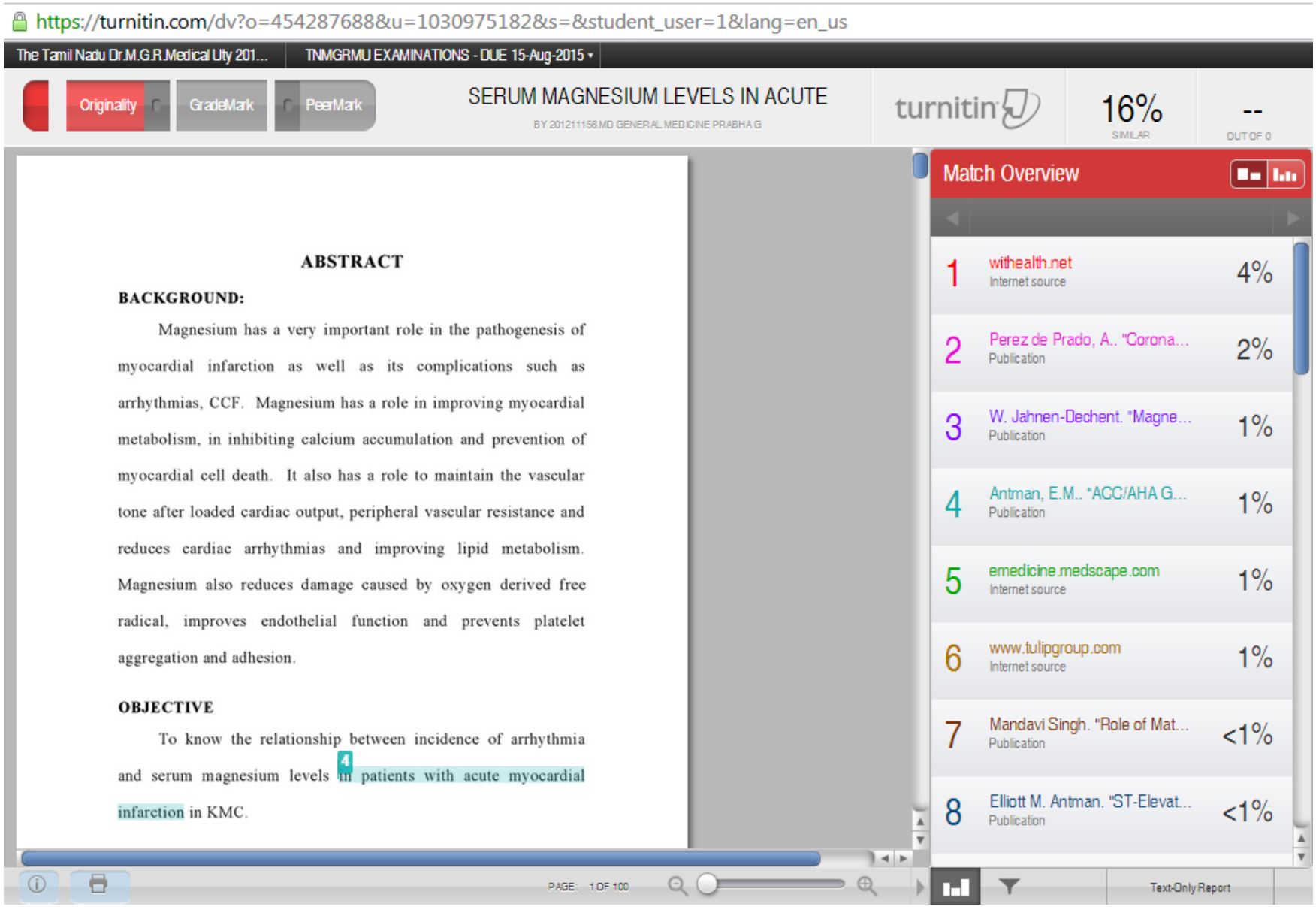
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ABSTRACT

BACKGROUND:

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OBJECTIVE

To know the relationship between incidence of arrhythmia and serum magnesium levels in patients with acute myocardial infarction in KMC.

METHODS

By using simple random methods 50 patients admitted in Cardiology Department of KMC with acute myocardial infarction was selected over a period of six months.

RESULTS

Significant difference have been noted in the incidence of arrhythmia between hypomagnesemics and normomagnesemics people in myocardial infarction.

CONCLUSION

Patients with low magnesium levels are prone to develop arrhythmias and risk factors like hypertension, diabetes increases the risk of arrhythmias in CAD patients.

KEY WORDS

Magnesium, myocardial infarction, arrhythmias.

INTRODUCTION

Since a long time we know that for normal growth and function of biological forms inorganic salts are required. Thus, Pasteur (1860) proved that yeast can grow only if the culture medium has inorganic compounds. In human body there is a tendency to maintain fluid balance both as a whole as well as between the three compartments.

This is achieved by intricate play of electrolytes, hemodynamics and other external forces. At present world, mineral metabolism is under rapid expansion. It has come to evidence that not only carbohydrates, proteins and fat, but also minerals are necessary to life. Now the significance of not only vitamins and other organic substances but also minerals is under investigation.

Magnesium has a very important role in pathogenesis of acute myocardial infarction as well as its complications like arrhythmias and CCF.[1-5]

Magnesium has a very important role in other cardiovascular diseases also.

Magnesium ions play a role in maintaining the functional integrity of the myocardium.

Investigations showed that magnesium levels are decreased in the initial phases of AMI and also the findings directly correlated with occurrence of complications like arrhythmias. There are evidences to show that myocardial magnesium concentration are found to be very low in patients with sudden cardiac death due to ischemia. The factors responsible for this are the role of magnesium in ventricular fibrillation which results in sudden death in IHD patients and coronary vasospasm due to magnesium deficiency[6-9]. The eighth most common element in earth is magnesium and is usually tied up with other mineral deposits such as magnesium carbonate, dolomite. The autonomic mass of magnesium is 24.305, specific gravity is 1.738 and boiling point is 1090 degree C.

It is a group II element in periodic table. Magnesium salt is soluble in water.

Role of magnesium is important for both plants and animals. Magnesium is the central ion of the chlorophyll and is a fourth most abundant cation in vertebrates.

Other uses are technical and medical applications like alloy production, fertilizers and healthcare.[10] Antacids and laxatives also utilizes magnesium salts.

REVIEW OF LITERATURE

Myocardial infarction is a syndrome as a result of injury to myocardial tissue due to imbalance between perfusion and demand. Main cause being coronary atherosclerosis with a superimposed coronary thrombus.

In 2007 expert consensus document redefined acute MI as detection of a rise and fall in cardiac troponin with at least one value above the 99^{th} percentile of the upper reference limit (URL) together with evidence of ischaemia.

Ischaemia can be either symptomwise, ECG changes of new ischaemia or development of pathologic Q waves in ECG or imaging evidence of infarction.

Included in this definition were sudden cardiac death and evidence of myocardial ischaemia (new ST elevation LBBB or coronary thrombus) and biomarker elevation > 3 x URL for post – PCI patients, 5 x URL-for post CABG patients.

Documented stent thrombosis was also included in this new definition.

Established MI was defined as anyone of the following criterion:

a) Development of new pathologic Q waves on serial ECGs

b) Imaging studies showing MI

c) Pathologic findings of healed or healing MI.

CLINICAL CLASSIFICATIONS OF DIFFERENT TYPES OF MI[11]

Type-I	Spontaneous MI related to ischemia from a coronary plaque rupture/dissection.
Type-II	MI due to ischaemia resulting from increased oxygen demand or decreased supply.
Type-III	Sudden cardiac death, symptoms of ischaemia, new ST elevation or LBBB or coronary thrombus.
Type-IV a	MI associated PCI
Type-IV b	MI associated stent
Type-V	MI associated CABG

CLINICAL DIAGNOSIS

If a patient is suspected to have chest pain of cardiac origin ECG should be taken within 10 minutes. If ECG has evidence of acute ST-segment elevation or new LBBB, emergent reperfusion treatment with PCI or fibrinolysis is indicated.

If patient has history suggestive of cardiac chest pain and no EGC criteria for reperfusion therapy then patient might have unstable angina or NSTEMI.

SIGNS AND SYMPTOMS

Crushing substernal chest pain described as squeezing or constriction pain which radiates to left shoulder associated with an impending sense of doom.

In postoperative patients, elderly and in diabetic MI can occur without chest pain.

If there is sudden pain which is tearing or knife like and radiates to back aortic dissection should be considered.

Associated Symptoms

Dyspnoea, diaphoresis, lightheadedness, fatigue, palpitations, acute confusion, nausea, vomiting and indigestion.

Gastrointestinal symptoms are common in IWMI.

Physical examination does not play a much role in MI but is important for risk stratification or to rule out other mechanical complications.

DIFFERENTIAL DIAGNOSTIC CONSIDERATIONS FOR ST-SEGMENT ELEVATION MYOCARDIAL

infarction

Comorbid ischaemia	ST elevation but no ischaemia	Chest pain but no ischaemia
Aortic dissection Systemic arterial embolism Hypertensive Crisis Aortic stenosis Cocaine use Arteritis	Early repolarization LVH LBBB Hyperkalaemia Brugada syndrome	Aortic dissection Myopericarditis Pleuritis Pulmonary embolism Costochondritis GI disorders

LABORATORY INVESTIGATIONS

Troponins – Troponin T and Troponin I are useful in the diagnosis and management of unstable angina and NSTEMI because of their high sensitivity, it can interpreted at bedside rapidly available universally helps in detection of myocardial necrosis.[12]

Troponin elevation in absence of IHD is seen in

a) congestive heart failure
b) HOCM
c) Aortic dissection
d) Cardiac contusion
e) Pulmonary embolism
f) Acute neurologic disease
g) Drug toxicity

CREATININE KINASE

Appreciable rise in CK levels takes 4-6 hours.

CK – CK-MB is elevated in pericarditis and myocarditis which may cause diffuse ST-segment elevation.[13]

MYOGLOBIN

Peak level is seen in 1-4 hours which allows diagnosis of acute MI.

There is lack of cardiac specificity which limits clinical utility of myoglobin.

Diagnostic testing includes ECG changes and echocardiography.

RISK STRATIFICATION

TIMI Risk Model for prediction of short-term mortality in ST-segment elevation MI patients.

HISTORY

Age	65-74 years	2 points
Age	> 75 years	3 points

Angina /DM/HTN 1 point

PHYSICAL EXAMINATION

HR > 100 bpm 2 points

SBP < 100 mgHG 3 points

Killip class II-IV 2 points

Weight < 67 kg 1 point

PRESENTATION

Anterior ST elevation/LBBB time to treatment > 4 h	1 point
Time to treatment > 4h	1 point

TIMI RISK SCORE = TOTAL POINTS (0-14)

a) TIMI score of 9 or more predicts 30day mortality of 35% approximately.

b) TIMI score of 0 or 1 has 30 day mortality rate less than 2%.

c) The predictors for poor prognosis are advanced age, HTN, Killip Class II-IV at presentation, DM, weight and time to treatment of more than 4 hours.

THERAPY

ST elevation MI is the most critical clinical condition in cardiology but also most satisfying when it is managed appropriately. Present availability of treatment measures can save many lives and helps to restore in normal cardiac function.

Cardiac Markers - reduced blood flow to the myocardium will lead to cardiac Ischaemia. When ischaemia is prolonged (20-40 minutes or more) it leads to Injury. Death of the injured myocardial cells is infarction and they release macromolecules which can be detected in the bloodstream. Cardiac markers are CK-MB isoforms, CK-MB, myoglobin and cardiac troponins I and T. CK-MB

Isoforms and cardiac troponins I and T are very specific to cardiac necrosis, and they can be detected only after 4-6 hours of MI.

HIGH RISK STEMI

a) ST elevation MI with any of the following features:

a. Previous history of MI

b. New LBBB

c. Ejection fraction less than 35%

d. Extensive ST elevation

e. Killip class > 2

b) Anterior wall MI with ST elevation more than 2mm in 2 or more leads

INDICATIONS FOR DES USE IN STEMI

1) Diabetes mellitus

2) Long lesion in culprit vessel

3) Smaller vessel size

4) Proximal occlusion of LAD

5) Patients ability to comply with prolonged double platelet therapy.

STRATEGY FOR REPERFUSION

It is indicated for all patients with history of chest pain for less than 12 hours and with ST elevation or new onset LBBB, any ECG evidence of ongoing ischaemia and symptoms for more than 12 hours. Reperfusion therapy with PCI should be considered in

stable patients with more than 12-24 hours of symptoms. In stable patients with no angina and PCI is not indicated after 24 hours.

ASSESSMENT OF REPERFUSION OPTION FOR STEMI PATIENT

Step-1:

Time and Risk assessment.

Time since the onset of systems.

Risk of STEMI.

Time required for transportation to a PCI lab.

Risk of fibrinolysis.

Step-2

To identify if fibrinolysis or invasive strategy is needed.

Within three hours of presentation and there is no delay for invasive procedure, both are equally beneficial.

Fibrinolysis is generally preferred under the following conditions:[14]

1) Presentation of less than 3 hours of onset of symptoms and there is a delay to invasive procedure.

2) Catheterization lab is not available or occupied, lack of accessibility to a PCI lab, any difficulty in vascular access.

3) Delay to invasive strategy either prolonged door to balloon time or medical contact to balloon or door to balloon is more than 90 minutes.

Invasive strategy is preferred under following conditions

1) Availability of skilled PCI lab with surgical facilities.

2) High risk from STEMI either cardiogenic shock or Killip class > 3.

3) Bleeding manifestations or ICH.

4) Late presentation more than 4 hours.

5) Situations were diagnosis of STEMI is doubtful.

GENERAL MEASURES

Routine measures include oxygen supplementation, analgesics like morphine 2-4

Mg IV which can be increased up to 8 minutes at 5-15 minutes of interval as per requirement.

Fibrinolysis: Absolute contraindications

1) Features of aortic dissection.

2) History of ICH

3) Ischaemic stroke less than three months of duration except for acute ischaemic stroke of less than 3 hours.

4) Any intracranial neoplasm.

5) Arteriovenous malformation.

6) Bleeding diathesis.

7) Head or facial trauma within three months.

RELATIVE CONTRAINDICATIONS

1) Uncontrolled hypertension.

2) Severe hypertension of more than 180 mmHg of SBP and DBP more than 110 mmHg. CPR or major surgery within less than three weeks.

3) Noncompressive vascular punctures.

4) Recent internal bleeding (2-4 weeks).

5) Prior history of any allergic reactions to streptokinase.

6) Active peptic ulcer.

7) Pregnancy.

8) Current use of anticoagulants: Higher the INR, more risk of bleeding.

FIBRINOLYTIC THERAPY DOSAGE

Streptokinase: 1.5 MU IV over 30-60 minutes.

Tenecteplase: If < 60, give 30 mg single IV bolus; if 60-69 kg, give 35 mg single IV

Bolus; if 70-70 kg, give 40 mg single IV bolus; if 80-89 kg, give 45 mg single IV

Bonus; if > 90 kg, give 50 mg single IV bolus.

Reteplase: 10 U IV over 2 minutes, repeat after 30 minutes.

ANTIPLATELET AND ANTITHROMBIN CO-THERAPIES ALONG WITH FIBRONYLSIS

Aspirin 125-325 mg of loading dose should be chewed by the patient.

Maintenance therapy 75 mg per day.

Clopidogril 300 mg- if age < 75 years loading dose. If age > 75 years-75 mg, maintenance dose of 75 mg per day should be given for a minimum period of one year.

Enoxaparin- Age <75 years and creatinine < 2.5 mg/ml should be given 30 mg bolus followed by 15 minutes later 1 mg/kg every 12 hours subcutaneous dosage until the time of discharge or for a maximum period of eight days.

Heparin- 60 units per kg IV bolus to a maximum of 4000 units followed by IV

Infusion of 12 units per kg to a maximum of 1000 units per hour.

aPT has to be maintained at 50-70 seconds and should be monitored at 3, 6, 12/24 hours.

Fondaparinux- 25 mg IV bolus followed by 2.5 mg s.c. its dose once daily up to eight days.

Nitroglycerin is available as sublingual tablets,IV form, aerosol spray.

INDICATIONS

1) Antianginals if ischaemic pain is suspected.

2) In acute phase of AMI, large AWMI, CHF, persistent or recurrent ischaemia, hypertension.

3) Beyond 48 hours doses continued for patients with recurrent angina and persistent pulmonary congestion.

4) In case of hypertension urgency.

PRECAUTIONS AND CONTRAINDICATIONS

It has to be limited in AMI patients to a fall of systolic BP by 10% in normotensive, 30% drop if hypertensive and avoid drop below 90 mmHg.Should not be mixed with other drugs. Patient have to lie down or sit while getting the medication. The aerosol spray should not be shaken as it affects the metered dose.

CONTRAINDICATIONS

1) Bradycardia.

2) Right ventricular infarction.

3) Hypotension.

DOSAGE OF IV INFUSION

12.5-25 mcg of IV bolus if no sublingual or spray given.

Infusion at rate of 5-20 mcg/min. In emergency IV is a route of choice. Titration of dose is important to achieve effects. Drug has to be diluted in D5 or NS.

Sublingual route - 0.3-0.4 mg of one tablet repeated every 5 minutes.

Aerosol spray 1-2 spray for 0.5-1 second at an interval of 5 minutes, maximum is 3 sprays within 15 minutes.

BETA-BLOCKERS

All myocardial infarction patients should be given the benefit of beta-blockers if there is no contraindication irrespective of primary PCI.

In case of tachyarrhythmia or hypertension IV beta-blockers is preferred.

CONTRAINDICATIONS FOR BETA-BLOCKERS

1) Low output state.

2) There is risk of cardiogenic shock.

3) Heart failure.

4) Others relative contraindications are prolonged PR interval, active asthma, any AV block, reactive airway disease.

DOSAGE

Esmolol 500 mcg/kg over 1 minute followed by 50 mcg/kg over 4 minutes as IV injection. Maintenance dose is at 60-200 mcg/kg/min infusion.

Metoprolol- 2.5-5 mg IV over 2 minutes can be given up to three doses.

Oral 50 mg 6th hourly for the first two days and then 100 mg 12th hourly has to be continued.

Atenolol- 1 mg/min as IV dose to a maximum of 5-10 mg.

Propranolol-0.15 mg/kg by IV route.

ACE INHIBITORS

All patients with anterior infarction should be given ACE inhibitors within 24 hours orally unless there is any contraindications.

DOSAGE

1) Captopril 6.25 mg initially, 12.5 in 2 hours followed by 25 mg at 10-12 hours, target dose is up to 50 mg twice a day.

2) Lisinopril- 5 mg initially up to 10 mg every day.

3) Ramipril-2.5 mg twice a day up to 5 mg twice a day if tolerated.

4) Valsartan- 20 mg up to 160 mg twice a day.

5) Losartan-12.5 mg initially followed by 50 mg daily.

6) Eplerenone-12.5 mg initially followed by 50 mg every day.

PRIMARY PCI

Should be performed in STEMI patients or with new LBBB if the patient can undergo PCI within 12 hours of symptoms, and balloon inflation within 90 minutes of presentation.

SPECIFIC CONSIDERATION:[15]

The goal of PCI is to be performed as quickly as possible with a medical contact- to-balloon or door-to-balloon of less than 90 minutes.

For symptoms less than 3 hours PCI should be performed within the first 1 hour, in new onset LBB patients with age less than 75 years, in pulmonary edema CHF patients within 12 hours onset of symptoms.

Rescue PCI is a procedure performed in a coronary artery that remains occluded in spite of fibrinolytic therapy and if there is less

than 50% resolution of ST segment elevation after 60-90 minutes of the start of fibrinolytic therapy.

Introcoronary medications during primary PCI.

Medications	**Dosage**
Nicorandil	500 mcg single bonus to a total of 10 mg
Eptifibatide	180 mcg/kg x 2 doses
Verapamil	200 mcg as a single bolus to a total of 1 mg
Epinephrine	50-200 mcg
Nitroprusside	100 mcg single bonus to a total of 1 mg
Titrofiban	10 mcg/kg (IC) followed by 0.15 mcg/kg/min
Streptokinase	250kU over 3 min
Adenosine	12 mcg as a single bolus to a total of 4 mg
Diltiazem	200 mcg as a single bolus to a total of 5 mg
Abciximab	0.25 mg/kg (IC) followed by 0.125mcg/kg/m for 12h (IV)
Nicardipine	200 mcg as a single bolus to a total of 1 mg IC: intracoronary IV: Intravenous

TIMI ANGIOGRAPHIC FLOW GRADING [16]

Epicardial blood flow	TIMI flow grades (TFG)
0	Absence of anterograde after occlusion
1	Contrast agent passes through occluded area without opacifying whole length of artery by the end of injection
2	Contrast agent opacifies the whole artery but is very slow in nonculprit arteries or proximal to occluded portion of the artery
3	Normal anterograde flow as in nonculprit artery or proximal to occlusion
4	Anterograde flow and clearance of the contrast is faster compared to nonculprit arteries

TIMI MYOCARDIAL PERFUSION GRADING[17]

Micro vascular flow	TIMI Myocardial Perfusion Grades (TMPG)
0	Absence or minimum blush of the myocardium in the distribution section of the culprit artery
1	Persistent myocardial blush; the contrast agent enters the microvasculature, but it does not normally pass to the venous phase:"persistent stain" is detected at the beginning of the next injection (> 30s)
2	Delayed blush and washout of the myocardium: the myocardial stain is evident (maximum level or minimum decline inintensity) by the end of the injection (3 heart beats for washout)
3	Normal blush: entry and exit of the contrast agent from the microvasculature at normal speed (total or high washout of the dye within 3 heart beats

COMPLICATIONS OF ACUTE MYOCARDIAL INFARCTION

1) ISCHAEMIC

   a. Reinfarction

   b. Angina

   c. Extension of the infarct

2) MECHANICAL

   a. Cardiogenic shock

   b. Heart failure

   c. Aneurysms/cardiac rupture

   d. Mitral valve dysfunction

3) ARRHYTHMIC

   a. Atrial arrhythmias

   b. Ventricular arrhythmias

   c. Sinus node dysfunction

   d. atrioventrcular node dysfunction

4) EMBOLIC COMPLICATIONS

a. Central nervous system or peripheral embolisation

5) INFLAMMATORY

a. Pericarditis

ISCHAEMIC COMPLICATIONS

a) Extension of the infarct is nothing but increase in the amount of myocardial necrosis within the infarct zone of myocardial infarction. Incidence of reocclusion of the artery is about 5-10% at the time of discharge and increases to 25-30% at one year.[44]

Hence such patients have poor prognosis.[45-46]

Reinfarction is usually seen in patients with diabetes mellitus or old CAD. With the advent of PCI and stent surgery reinfarction rate has substantially decreased to about 3% during first 90 days following MI.[47]

PATHOPHYSIOLOGY

When the infarcted artery reoccludes it leads to reinfarction but reocclusion of the infarcted artery does not always lead to reinfarction due to abundance of collateral circulation. Reocclusion

following post fibrinolytic therapy is 5-10% and has a poor prognosis. When PCI is done the rate is much lower.

The pathophysiology of post infarction angina is plaque rupture similar to unstable angina and the management is same as unstable angina. Echocardiography is a diagnostic method of choice.

SIGNS AND SYMPTOMS

1) Continuous chest pain

2) Elevated creatinine kinase level

TREATMENT

Pharmacological therapy with heparin, nitroglycerin, beta blockers and statins are usually given to patients who have had MI and are presently experiencing ongoing ischaemic symptoms.[48]

Depending on clinical situation antiplatelet therapy can be given and intraaortic balloon pump is inserted promptly in haemodynamically unstable patients.

EMBOLIC COMPLICATIONS

Incidence of embolism is less than 2% and maximum incidence is with AWMI patients. Large AWMI may have up to 60% incidence of mural thrombus.[49-50]

PATHOPHYSIOLOGY

Most commonly emboli originates from left ventricle and the cause being wall motion abnormalities or aneurysms.

Atrial fibrillation along with ischaemia can lead to systemic embolisation.

SIGNS AND SYMPTOMS

Stroke is the most common clinical presentation. Other features are limb ischaemia, mesenteric ischaemia or renal infarction. Usually systemic embolic occurs within 10 days of acute myocardial infarction. Limb ischaemia presence with cold clammy painful pulseless extremities. Features of renal infarction are pain in the flank, haematuria and raising urea creatinine.

Mesentric ischaemia presence with severe abdominal pain, bloody diarrhea and anorexia.

MANAGEMENT

Heparin through IV route should be given immediately in the absence of any active bleeding and the target APTT time should be 50-70 seconds and heparin has to be continued until Warfarin gets into therapeutic action.

Warfarin has to be continued for at least 3-6 months with INR maintain at a range of 2-3. Patients with large akinetic areas detected by echo should also be given Warfarin for 3-6 months.

ARRHYTHMIAS IN MI

Literature shows that 90% of people with MI develop some of arrhythmia either immediately or later.

Arrhythmias occur within 24 hours in 25% of patients. Risk of ventricular fibrillation is more with STEMI than NSTEMI most peri-infarct are complications of myocardial infarction broadly classified into arrhythmia complications.

Sinus tachycardia – causes increased sympathetic activity which may lead to transient HTN or hypotension, tachycardia increases the diastole length and reduces coronary flow which leads to worsening of myocardial ischaemia.

Causes of persistent tachycardia are

1) Pain

2) Anxiety

3) Heart failure

4) Hypoxia

5) Hypovolemia

6) Pericarditis

7) Pulmonary embolism

8) Anaemia

In AMI patients treating sinus tachycardia plays an important role.

Treatment includes:

Analgesics, diuresis for heart failure, oxygenation, volume repletion for hypovolemia, beta blockers for ischaemia, anti-inflammatory measures.

PAC - may be a preceding event of SVT, atrial fibrillation or atrial flutter.

No specific therapy is indicated.

Pathogenesis is inflammatory associated with pericarditis or atrial distention due to enhanced left ventricular diastolic pressure.

PAROXYSMAL SVT

Incidence is less than 10%.

Management: Adenosine can be used if there is no hypotension.

In the absence of heart failure/hypotension synchronized electrical cardioversion is needed.

Atrial flutter - occurs in 5% or less usually it is temporary and is due to sympathetic overstimulation of atria.

Atrial fibrillation - incidence in 10-15% in acute myocardial infarction patients. During initial hours of MI cause of AF is LV failure, RV infarction ischaemic injury to atria. Other causes of AF in MI are pericarditis, other condition which increases LA pressure.

AF in MI increases the risk of stroke, mortlity or patients synchronized electrical cardioversion in the treatment.

Other modalities of treatment are IV Amiadarone, Digoxin, Beta blocker, if no hypotension Diltiazem.

Atrial fibrillation and atrial flutter increases risk of thromboembolism hence anticoagulation therapy with Heparin is needed if no contraindication.

Accelerated junctional rhythm is most common in IWMI caused by increased automatically of junctional tissue.

Treatment is to correct the underlying ischaemia.

BRADYARRHYTHMIAS

A) Sinus bradycardia is common in inferior and posterior wall infarction.

Pathogenesis is stimulation of cardiac vagal afferent receptors leading to efferent cholinergic stimulation of the heart which results in bradycardia and hypotension.

Treatment is not urgent in case of isolated sinus bradycardia with heart rate > 40 bpm.

Treatment is mandatory with atropine sulfate 0.5-1 mg every 3-5 minutes to a maximum of 0.03-0.04 mg/kg if HR <406 pm.

If atropine is ineffective additional treatment with dopamine 5-20 mg/kg/min IV, dobutamine or epinephrine is used.

External pacemaker is needed for resistant bradycardia.

B) Junctional bradycardia - is a protective phenomenon with a rate of 35-60 bpm in IWMI patients. No specific treatment is needed.

First degree AV block- prolongation of PR interval of more than 0.2 seconds is the feature in ECG.

Incidence is 15%, and most common in IWMI patients. Treatment is unnecessary unless there is hemodynamic compromise.

Atropine is the treatment of choice for first degree AV block with hypotension.

Second degree AV block - Mobitz type 1 is associated with narrow QRS complex and his most often associated with IWMI. Incidence is around 10% in acute myocardial infarction patients. Treatment is rarely required.

Mobitz type 2 AV block is associated with poor prognosis as it has tendency to progress to complete heart block. A temporary or permanent pacemaker must ultimately be placed.

Third degree AV block or complete heart block - occurs in 15% of patients with MI. It may occur either with AWMI or IWMI patients. The development of block with inferior infarction is gradual progressing from first degree or type 1 second degree block.

The level of block in most patients is supranodal or intranodal and escape rhythm is usually stable with QRS complex being narrow and rate more than 40 bpm.

In 30% of patients the level of block is below Bundle of Hiss which results in escape rhythm and heart rate less than 40 bpm and a broad QRS complex.

IWMI with complete heart block usually responds to Atropine and in most patients the block resolves spontaneously within few days. Prognosis of patients with inferior wall MI with complete heart block is good compared to AWMI patients with CHB as they may require permanent pacemaker.

Intraventricular blocks **-** conduction from Bundle of His is transmitted through the anterior division of the left bundle, the posterior division of left bundle and the right bundle. An abnormality of electrical conduction in any of these bundle is seen in 15% of AMI patients.

Isolated left anterior fascicular block incidence is about 3-5% and rarely progresses to complete heart block. The blood supply of right bundle branch is from left anterior descending artery. Hence in about 2% of AMI patients new RBBB is seen.

Rarely it progresses to complete heart block. In anterior MI patients with a new RBBBthe cause of death is mostly cardiogenic shock.

Bifascicular block is a combination of RBBB with LAFB usually occurs with occlusion of proximal LAD. There is more chance of development of complete AV block. Bifascicular block with first degree AV block is known as trifascicular block. 40% patients may progress to complete heart block.[18]

VENTRICULAR ARRHYTHMIAS

PREMATURE VENTRICULAR CONTRACTIONS

Previously PVCs were considered as indicators of impending ventricular arrhythmias but these days VPCs usually occur in MI patients without any further progression to arrhythnmias. Prophylactic treatment is not indicated instead we should aim to correcting any electrolyte of metabolic abnormalities as well as identifying and treating recurrent ischaemia.

Accelerated idioventricular rhythm is seen in about 20% of AMI patients.

ECG is characterized with a wide QRS complex with a regular escape rate which is faster than atrial rate but less than 100 bpm. They are short and transient and terminate spontaneously. Mechanism involves structural damage of SA node or AV node and depresses nodal automaticity. An abnormal ectopic focus in the ventricle is other mechanism. Prognosis is not affected by the

presence of accelerated IVR. There is no need for temporary pacing or antiarrhythmic drugs as it can result in clinically significant bradycardia or asystole hence an accelerated IVR should not be treated.

Nonsustained ventricular tachycardia is defined as three or more consecutive ventricular ectopic beats with a rate more than 100 bpm and should last less than 30 seconds.

MECHANICAL COMPLICATIONS Of acute myocardial infarction are

1) Ventricular free wall rupture

2) Ventricular septal rupture

3) Papillary muscle rupture with severe MR

These complications can lead to cardiogenic shock.

VENTRICULAR FREE WALL RUPTURE (VFWR)

VFWR is most critical complication of AMI as the important cause of death after LV failure and also 15-30% of death in AMI is due to VFWR.[19-20] VFWR can be associated with large transmural infarction and also antecedent infarct expansion.

The most serious complication of VFWR is acute haemopericardium and cardiac tamponade which can result in death.

The incidence of VFWR 0.8-6.2%.

The incidence has declined in recent years due effective blood pressure control measures, effective reperfusion therapy availability ACE inhibitors, beta-blockers, and reduced use of heparin.

RISK FACTORS FOR VFWR ARE

1) Age more than 70 years

2) Female sex

3) Newly diagnosed MI

4) Q waves on ECG

Therefore during acute phase of STEMI patients receiving fibrinolytic therapy later than 14 hours of onset of STEMI, in contrast patient who has history of angina pectoris in the past, multivessel coronary disease/chronic heart failure are less widely to develop VFWR due to presence of collaterals and ischaemic preconditioning.[21-22]

CLINICAL FEATURES

It depends on acuity of onset, location and size of rupture.

Patients present with severe chest pain, hypotension, haemodynamic Instability, asystole or death.

In subacute course pateints present with hypotension, arrhythmias, syncope, shock or recurrent chest pain.

THREE TYPES OF VFWR

Type-1 Slit like tear usually associated with anterior infarcts and occurring with 24 hours.

Type-2 At the border between infarcted and viable myocardium. There is an erosion of infarcted myocardium.

Type-3 Early aneurysm formation seen in old and severely expanded patients.

DIAGNOSIS OF VFWR

Early diagnosis and early treatment as necessary for survival. A high index of suspicion is essential.

Echo is the choice of diagnosis. Diagnostic feature in echo are moderate to large pericardial effusion and impending pericardial tamponade.

TREATMENT OF VFWR

Prevention is by early reperfusion therapy or percutaneous coronary Intervention. The best of choice is early surgical repair after haemodynamic stability.

IV fluids, ionotropic agents and pericardiocenters are emergency treatment.

Surgical techniques include intraaortic balloon pump, infarctectomy, biologic glue patches with polythylene terephthalate polyester fiber, Teflon and use of pledgeted stures.

The mortality rate is very high and it greatly depends patients preoperative hemodynamic status.

VENTRICULAR SEPTAL RUPTURE

This is rare but most life threatening complication of AMI. Inspite of appropriate medical and surgical therapy mortality rate is significantly high.

In olden ear i.e., prethrombolytic era incidence was 1-3%, 1 at present, incidence is 0.2-0.34%[23-24.]

There is bimodal distribution with peak in first 24 hours and initial. Second peak on days 3-5 and rarely after 2 weeks of AMI.

Risk factors for ventricular rupture are

1) Old age

2) 65 years

3) Female sex

4) Poor septal collateral circulation

5) Single vessel disease

VSR can be classified into simple and complex.

If the perforation is at same level on both sides of the septum with a direct through and through communication as simple ventricular septal rupture.

Complex septal rupture is one with extensive hemorrhage and irregular serpiginous traits.

Septal ruptures are mostly seen in anterior MI with occlusion of LAD artery.

ECG changes in these include ST-segment elevation, Q waves in II, III, avF.

CLINICAL FEATURES

Chest pain, dyspnea, hypotension, LV failure, and shock.

On examination, patient will have a holosystolic murmur. The murmur is best heard at lower left sternal border, other findings are palpable parasternal systolic thrill, S3 gallop.

In patients who present with cardiogenic shock with VSR murmurs cannot be appreciated. In contrast patients with acute MR may have soft systolic murmur in apex with no thrill.

DIAGNOSIS OF VENTRAL SEPTAL RUPTURE

Echocardiography, colour flow Doppler are the imaging modalities of choice uses of imaging technique as to assess ventricular function, to localize the site and size of septal rupture, to calculate RV systolic pressure and to quantify left to right shunt.

Confirmation can be done by cardiac catheterization.

MANAGEMENT OF VSR

Prompt diagnosis and immediate treatment is necessary. These steps in order has to be followed haemogynamic stabilization, angiography surgery.

Supportive measures like nasal oxygen, ventilator support, diuretics, vasodilators , ionotropic agents should be used.

Medical management is only temporary to stabilize patients before surgery.

Current guidelines from the American College of Cardiology is emergency surgical repair irrespective of patients clinical condition.[25-28]

Surgical measures are hypothermic and cardiopulmonary bypass, removal of all friable margins of septum and necrotic area followed by reconstruction of ventricular walls and septum. Latest technique which is being tried in some institution are percutaneous closure of septal rupture, datas about outcome of this method is still unavailable.

Repetition of surgical repair is done in patients with pulmonary systemic traction more than 2 residual septal defect

following surgery is seen in 28% and the mortality rate is very high.

Papillary muscle rupture with mitral regurgitation - One of the common complication of AMI is MR as a result of LV remodeling and it as an

Independent risk factor for death[29-32]. The usual period of occurrence of MR is 7-10 days following AMI where rupture can occur within 1-14 days. When MR is mild/moderate it is detected in routine echocardiography following MI and rarely results in haemodynamic compromise.

In contrast acute severe MR due to rupture of chordae tendinae/papillary muscles needs prompt surgical intervention as it can results in cardiogenic shock, haemodynamic instability and death.

Incidence of MR in AMI patients is 1% and usually involves the posteromedial papillary muscle due to single blood supply as compared to anterolateral papillary muscle as it has double blood supply.[33-35]

Clinical features of mild to moderate MR are mostly asymptomatic, in acute severe MR patients present with dyspnea,

fatigue, shock. On physical examination early to mid systolic murmur will be heard and is best heard at left lower sternal border. S3 and S4 gallop may be present.

DIAGNOSIS

High index of suspicion is needed in patients who present with cardiogenic shock, pulmonary edema or new systolic murmur at the apex. Other imaging modalities are echocardiography, colour flow Doppler.

MANAGEMENT

To stabilize the patient haemodynamically, to find out exact mechanism of MR are very essential for a good outcome. Pharmacological therapy includes diuretics, ionotropic agents nitrates. In haemodynamically unstable patients aortic balloon counterpulsation is the treatment of choice.

In case of papillary muscle rupture resulting in MR emergency surgical intervention is needed. Either mitral valve repair, or mitral valve replacement has to be done.

In patients presently intermittent MR as a result of recurrent ischaemia.

There is no need for emergency surgery. Treatment for such patients is revascularization either by angioplasty or coronary artery bypass grafting.

LEFT VENTRICULAR ANEURYSM

An abnormal outward bulging and deformation of a localized area of myocardium during both systole and diastole is known as left ventricular aneurysm.

The incidence is 3-15% in AMI patients. Risk factors are female sex, single vessel disease, occlusion of LAD and absence of any previous heart disease.

The commonest location of LVA is aterolateral wall and usually occur with occlusion of LAD. LVA is composed of fibrous scar.

DIAGNOSIS

Physical examination may reveal third of fourth heart sounds, enlarged cardiac silhouette in chest x-ray.

ECG shows ST elevation, echo is 93% sensitive and 94% specific in diagnosis of LVA.

TREATMENT

Conservative management with close follow up for small or clinically insignificant aneurysms

Pharmacotherapy includes ACE inhibitors and anticoagulation for patients with left ventricular thrombus, and severe LV dysfunction.

Indications for surgery are ventricular tachyarrhythmias refractory to medical treatments, severe heart failure, recurrent thromboembolism.

Other miscellaneous complications of acute myocardial infarction are post

MI syndrome **(Dressler syndrome)**. In this present era, incidence of post MI syndrome has reduced compared to olden days that is before the advent of reperfusion therapy.[36-37] Dressler syndrome is considered to be an autoimmune process. Clinical features are fever, chest pain. Treatment measures are hospitalization and observation to look for development of cardiac tamponade. Other measures are rest, steroids, NSAIDS.

PERICARDITIS

Inflammation of the pericardial tissue which overlies infarcted myocardium is known as pericarditis. Clinical manifestation are severe chest pain of pleuritic in nature. ECG shows diffuse ST segment elevation[38]. Treatment measures are aspirin, NSAIDS.

Left ventricular mural thrombus - is a complication usually associated with anterior infarcts. Incidence ranges from 20-40%. Patients with LVMT are at high risk of developing systemic embolization.

Anticoagulation therapy is the treatment of choice. LVMT formation is affected by LV regional wall ischaemia, inflammation etc. Echocardiography is the imaging modality and choice. Pharmaceutical therapy includes Heparin followed by warfarin.

MAGNESIUM

Magnesium is a cofactor for many enzymatic reactions and plays a very important role in many physiological functions. Though magnesium is one of the important elements it is most neglected one. Hypomagnesemia is more important than hypermagnesmia.

Serum magnesium measurement is the most easy and affordable method although it does not reflect total content of magnesium in the body. In spite of having similar charge and chemical reactivity as calcium. It has antagonistic behavior. Total magnesium content in the body is 20 mmol/kg of fat free tissue[39]. Average magnesium in an adult of about 70 kg will be 1000 to 1120 mmol. Bone has 99% of the total magnesium remaining is present in the skeletal muscles. Hence bone and skeletal muscles provides reservoir of exchangeable magnesium to maintain physiological extracellular magnesium levels.

Intracellular magnesium is about 5-20 mmol/l and 1-5% is ionized and the rest is bound to proteins and ATP. 1% of the total magnesium is in the extracellular compartment, mainly in serum and RBCs.

Serum magnesium can be free or ionized and bound to anions like phosphate, bicarbonate, sulfate, citrate or proteins. Ionized magnesium has maximum biological activity.

Magnesium is usually intracellular and it acts as a cofactor for more than 300 enzymatic reactions mainly ATP synthesizing reactions. Magnesium plays a role in the following reactions -

glucose utilization, synthesis of proteins, fat, coenzymes and nucleic acid. Hence variation in magnesium levels can affect all these reactions. As a result magnesium plays a role in normal neurological function, muscle contraction and relaxation, neurotransmitter release, to regulate vascular tone, to maintain heart rate and helps in bone formation.[41]

REQUIREMENT OF MAGNESIUM

Humans have to include magnesium in diet regularly to prevent magnesium deficiency. Recommendation of Institute of Medicine is 310-360 mg for adult woman and 400-420 mg per day for adult men and 355 mg in pregnancy and lactation. Drinking water contains 10% of our daily magnesium intake and it acts a major magnesium source for humans[43].

Other source of magnesium are nuts, unprocessed cereals and seeds, fruits, legumes and meat. Dairy products are low in magnesium content.

MAGNESIUM ABSORPTION AND EXCRETION

Magnesium concentration is maintained by bone, kidneys and intestine.

Magnesium is mainly absorbed in small intestine 24-76% of magnesium is absorbed in the gut and the rest is eliminated in faeces. Kidneys also play a important role in maintaining magnesium homeostasis. Glomeruli filters 2400 mg of magnesium present in the plasma. 95% is reabsorbed immediately and remaining 3-5% was excreted via urine.

Major reabsorption site for magnesium is thick ascending limb of Loop of Henle.

MAGNESIUM STATUS ASSESSMENT

There are three methods, extracellular fluid has only 1% of total body magnesium. First method is serum magnesium estimation. As only 0.3% of total body magnesium is found in serum. It is not a good indicator of magnesium levels. Haemolysis has to be avoided as magnesium content is high in RBCs.

Second method is 24 hours excretion in urine - the test is little cumbersome and not much reliable. It requires 24 hours and is useful to assess difference between magnesium wasting by kidneys and medicines.

Renal wasting is indicated by high urinary excretion and low values indicate decreased intake or absorption.[43]

Third method is loading test/magnesium retention test - may serve for identification of patients with hypomagnesmic and normomagnesemic magnesium deficiencies. Retention of magnesium following acute oral or IV administration helps in assessing chronic loss. Magnesium is retained in bone hence decrease in bone content will cause higher retention in this test. It is a sensitive index of magnesium deficiency. Recent method is isotropic analysis of magnesium.

PATHOPHYSIOLOGY

Serum magnesium estimation and collection of 24 hours urine are the methods to detect hypomagnesemia. Normal magnesium concentration is 1.5 mg/dl. Hospitalized patients are at higher risk of developing hypomagnesemia.

Medications causing hypomagnesemia are Cisplatin,

Digoxin, aminoglycoside, Lasix. Patients with low magnesium levels have poor prognosis, requires more days for weaning from ventilators and high mortality rate. Causes for hypomagnesemia are poor intake, GI loss, diarrhea, malabsorption. Other causes include RTA, DM, hyperthyroidism, hypercalcemia and diuretics use. Chronic hypomagnesemia is difficult to diagnose

as it has only very little negative equilibrium. Causes are myocardial infarction, atherosclerosis, hypertension, premenstrual syndromes, kidney stones and psychiatric disorders.

CLINICAL FEATURES

They are nonspecific. Features are muscle fasciculation, tremors, depression, agitation, arrhythmias and other electrolyte abnormalities like hypokalaemia. Other features are loss of appetite, fatigue, nausea, vomiting and weakness. As magnesium level decreases features like numbness, tingling sensation, cramps, involuntary muscle contractions and seizures can occur.

Hypermagnesemia - Here again kidneys plan important role. In chronic kidney disease the compensatory mechanism will start to fail and results in high magnesium levels. Causes include excessive intake, antacids or laxative use. Incidence varies from 5.7-8%.

Clinical Features - slight elevation may cause hypotension, nausea, vomiting, cutaneous flushing, bradycardia, QT prolongation ECG and absence of tendon reflexes.

TREATMENT OF HYPO AND HYPERMAGNESEMIA

In stable patients with hypomagnesemia oral administration is the choice. IV administration of magnesium is used in acute conditions like ventricular arrhythmias. Treatment of hypermagnesemia is to discontinue the offending drug and severe hypermagnesemia is an indication for haemodialysis.

USES OF MAGNESIUM

Magnesium has an important role in IHD pathogenesis,and others like CAD, coronary artery spasm, atherosclerosis, coronary artery thrombosis, hypertension, chronic heart failure and arrhythmias.

INDICATIONS FOR MAGNESIUM SUPPLEMENTATION

1) Previous history of heart attack

2) Risk of developing ventricular arrhythmia

3) Systemic hypertension - has direct relationship between low magnesium levels and high BP.

4) CCF patients

5) Diabetes

6) Dyslipidemia

7) Ischaemic heart disease

8) Coronary artery spasm/thrombosis

9) Cardiac myopathy

10) Digoxin related arrhythmias

11) Torsedes de pointes

12) On patients with diuretic supplementation

13) Normal healthy patients without any coronary renal disorder or hypertension can be given magnesium supplementation to get sufficient magnesium insurance.

The best thing is to have diet rich in fresh fruits and vegetables with nutrient supplementation in order to maintain good magnesium stores and protect ourselves from cardiac conditions.

AIMS AND OBJECTIVES

1) To know the relation between level of serum magnesium and incidence of arrhythmias in patients with acute myocardial infarction who are presenting within 12 hours of onset of symptoms at Govt kilpauk Medical College & Hospital.

2) To assess how the variables like

a. Diabetes

b. Chronic kidney disease

c. Dyslipidemia

d. Low ejection fraction affects magnesium levels in myocardial infarction patients.

METHODS AND MATERIALS:

Study Group : All cases of acute myocardial infarction admitted in KMCH satisfying the case definition

Study design : Cohort study

Duration of study : 6 months

Conflict of interest : Nil.

Inclusion criteria :

Those patients presenting to hospital within 12 hours of onset of symptoms of angina.

Patients were considered to have acute myocardial infarction if they have any of the following criteria:

1) ECG changes of acute myocardial infarction.

2) Rise of cardiac enzymes.

Exclusion criteria :

Patients with hypokalemia.

METHODOLOGY

- The data of each patient was collected in the specific proforma which includes the name, age, sex.
- Each selected patient was subjected to detailed history and thorough physical examination.
- ECG was taken immediately after admission and everyday during the hospital stay.
- Investigations like Urea, Creatinine, Sodium, Potassium was done on the day of admission.
- Fasting lipid profile was done the next day.
- Serum Magnesium levels were assessed within 24 hours of admission and on the day of discharge/fifth day.
- Fasting blood sugar status was assessed everyday in diabetics.
- 2D Echocardiography was also done during the in-patient stay.

METHOD OF SERUM MAGNESIUM ESTIMATION

Blood samples of all the fifty patients selected was collected immediately after admission and serum was separated and magnesium analysis was done using magnesium kit (**CALMAGITE METHOD**) and on fifth day.

PRINCIPLE

This method helps to determine magnesium in serum, urine and CSF.

Magnesium combines with calmagite in an alkaline medium to form a red coloured complex. Any interference by calcium and proteins is eliminated by adding specific chelating agents or detergents. The intensity of red colour formed is directly proportional to the level of magnesium contained in the sample.

REFERENCE VALUES

Serum – (children 1.5-2 mEq/l, adult 1.5-2.5 mEq)

CSF- 2-3 mEq/l

Urine - 6-8 mEq/24 hours

CONTENTS IN THE KIT

Contents	25 ml	75 ml
L1: Buffer reagent	12.5 ml	37.5 ml
L2: Colour reagent	12.5 ml	37.5 ml
S: Magnesium standard	2 ml	2 ml (2 mEq/l)

The contents are stable at 2-8 degree C and the reagents are ready to use. Magnesium in serum or plasma is stable for seven days at 2-8 degree C.

PROCEDURE

Wavelength or filter: 510 mm/green.

Temperature: Room temperature

Light path: 1 cm

Pipette into clean dry test tube labeled as blank (B), standard (S), and test (T)

Addition Sequence	**B (ml)**	**S(ml)**	**T (ml)**
Buffer reagent (L1)	0.5	0.5	0.5
Colour reagent (L2)	0.5	0.5	0.5
Distilled water	0.5	0.5	0.5
Magnesium standard (S)	0.01	–	–
Sample	0.01	–	–

The samples are mixed well and incubated at room temperature for 5 minutes and the measurement of absorbance of the standard and test sample against the blank was measured within 30 minutes.

CALCULATIONS

Magnesium in mEq = Abs.T/Abs.S x 2

This procedure is linear up to 10 mEq/l.

Note: All precautions were taken to clean the test tubes used with 1% Hcl and were deionized before use. All the chelating agents and EDTA were removed completely before using the test tubes.

STATISTICAL ANALYSIS

The present chapter is based on the analysis of data and interpretation and discussion ofresults. However valid, reliable and adequate the data may be, it does not serve and purposeunless the data is carefully processed, systematically classified, scientifically analyzed,properly interpreted and rationally concluded.

After the data had been collected, it was processed & tabulated directly in to SPSS 17.0 software. SPSS version 17.0 statistical software was used and the results obtained thereby have

been analyzed and interpreted. These have been done on Age, gender, SHT, DM, CAD, Smoker, Alcoholic, Cholesterol, TGL, Arrhythmia Days, Arrhythmia Type, AWMI, IWMI, LWMI, ANGI, Magnesium level at day one and day five.

The purpose of the study isto find out the tendency of each parameter and also intends to find out the differences in the magnesium level in repeated observations, association of the arrhythmia with demographic, risk factor and Diagnosis variables namely, gender, age, SHT, DM, CAD, Smoking, Alcoholic, AWMI, IWMI, LWMI and Angi.

The following statistical techniques were used for analyzing the data

1) Descriptive statistics – Frequency, Means, Standard Deviation and Percentages.

2) Multiple responses tabulation was made to capture the frequency and combination of more than one diagnosis in the patients.

3) Paired t statistics was used to find out difference between the level of magnesium in the Day 1 Mg mEq/L and Day 5 Mg mEq/L among the patients.

4) Chi- Square test was used to find association between Presence of Arrhythmia and demographic profile; similarly with the Risk Factors and Diagnosis.

5) Independent t Statistics was performed to find the significance between presence and absence of Arrhythmia in several factors.

RESULTS

Descriptive Statistics

Parameter	N	Mean	SD	(Max - Min)
Age	50	54.4	12.4	(80 - 25)
Cholesterol	50	194.5	26.8	(262 - 128)
TGL	50	142.5	24.8	(199 - 78)
Arrhythmia Days	32	1.8	0.9	(1 - 4)

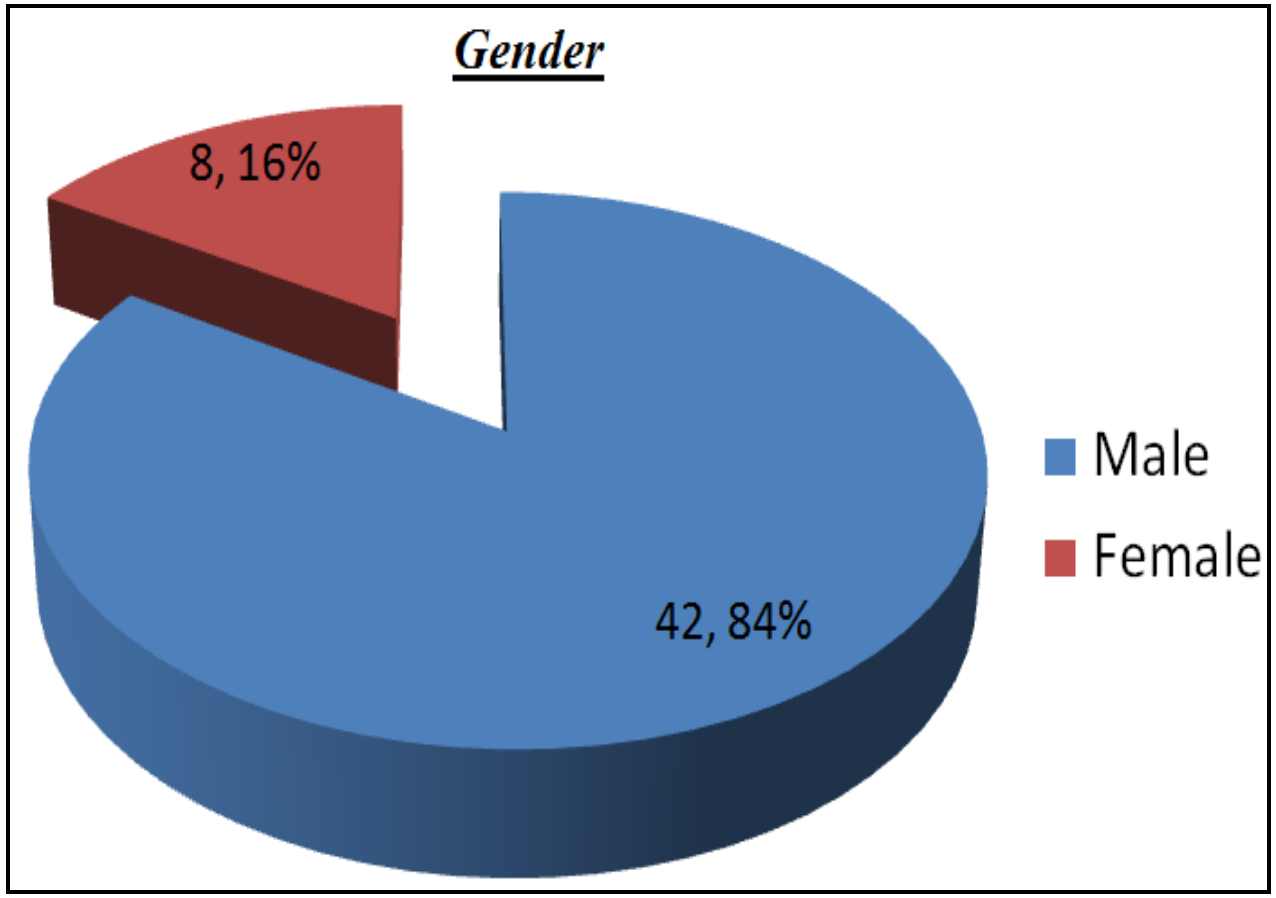
Of the 50 patients selected the age deviation was from minimum of 25 years and a maximum of 80 years with a mean of 54.4 and a standard deviation of 12.4.

32 patients out of 50 had arrhythmias in the course of hospital stay from day 1 to day 4 with a mean of 1.8 days with a standard deviation of 0.9. Cholesterol and triglyceride levels were estimated which range from maximum to minimum of 262 to 128 and 199 to 78 respectively.

Descriptive Statistics

Parameter	n	%
Gender	50	
Male	42	84.0%
Female	8	16.0%

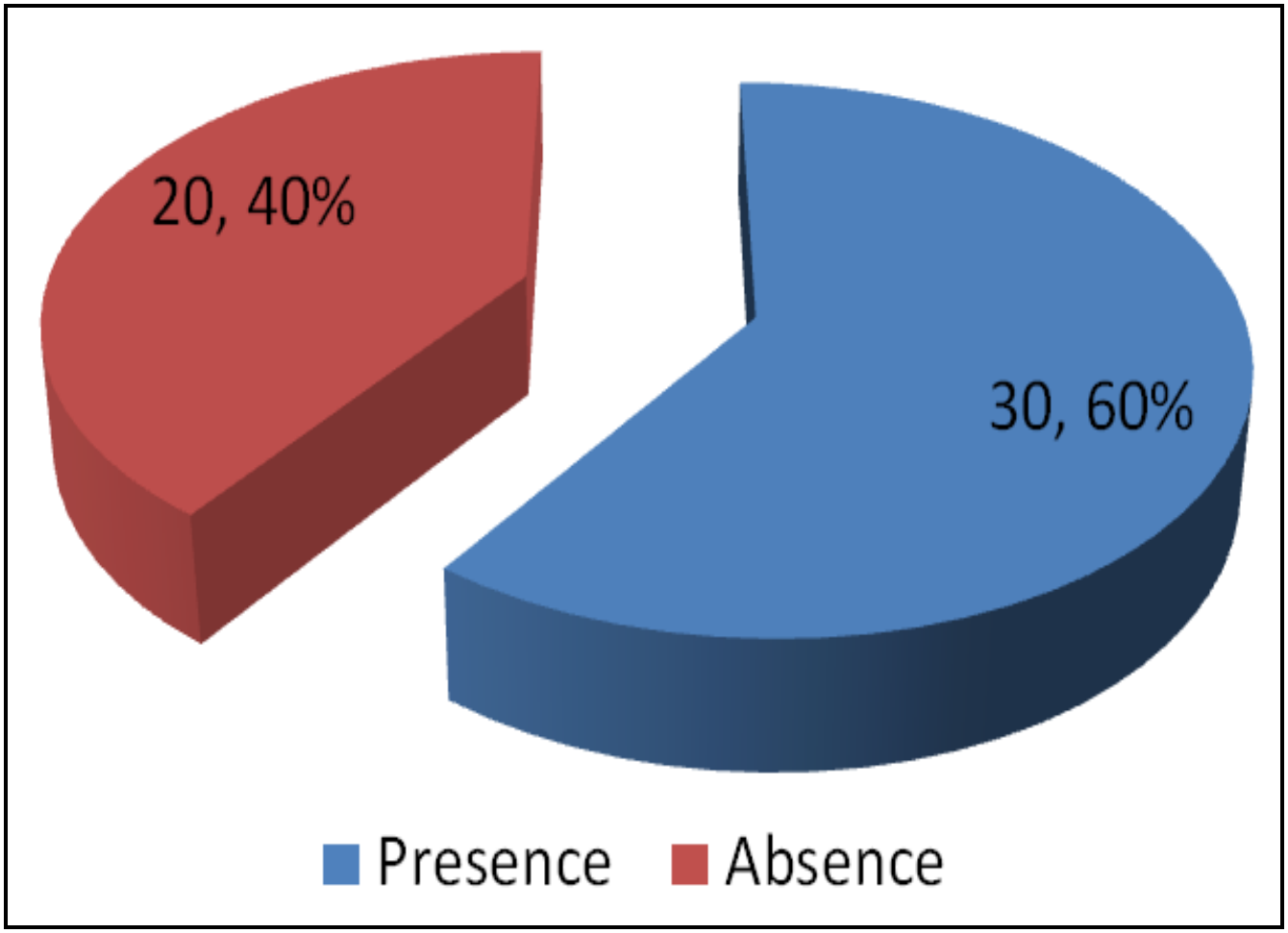
Total people under the study were 50 of which 42 are male (84%) and 8 were female (16%).


Gender
8, 16%
42, 84%
Male
Female

Descriptive Statistics

Parameter	n	%
SHT	50	
NO	20	40.0%
YES	30	60.0%

30 (60%) out of 50 patients had the risk of hypertension and 20 (40%) were not a known hypertensive.

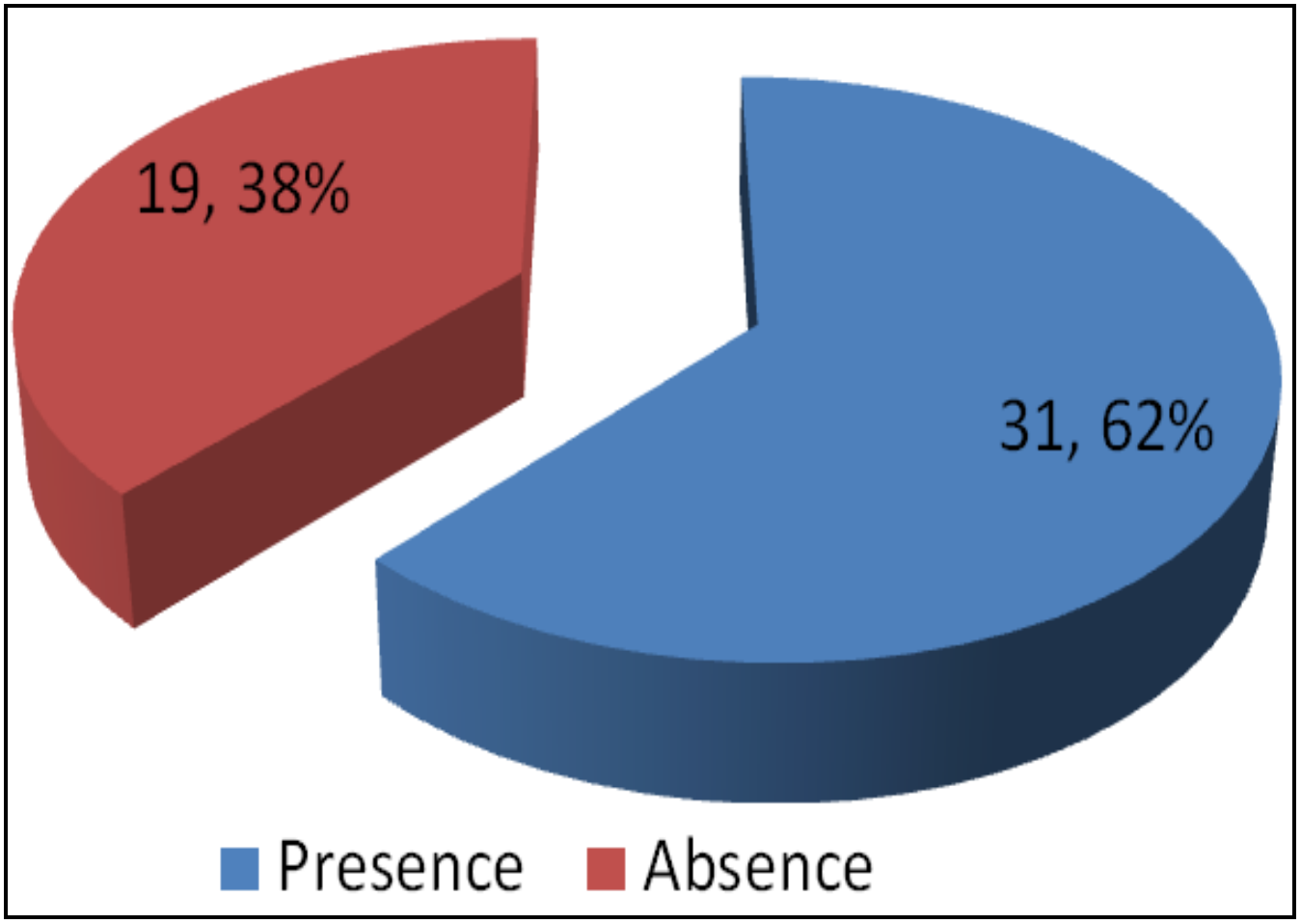

20, 40%
30, 60%
Presence
Absence

Descriptive Statistics

Parameter	n	%
DM	50	
NO	19	38.0%
YES	31	62.0%

31 (62%) out of 50 patients were known diabetic, 19 (38%) were nondiabetics in the study and the distribution is shown in the diagram above.

DM

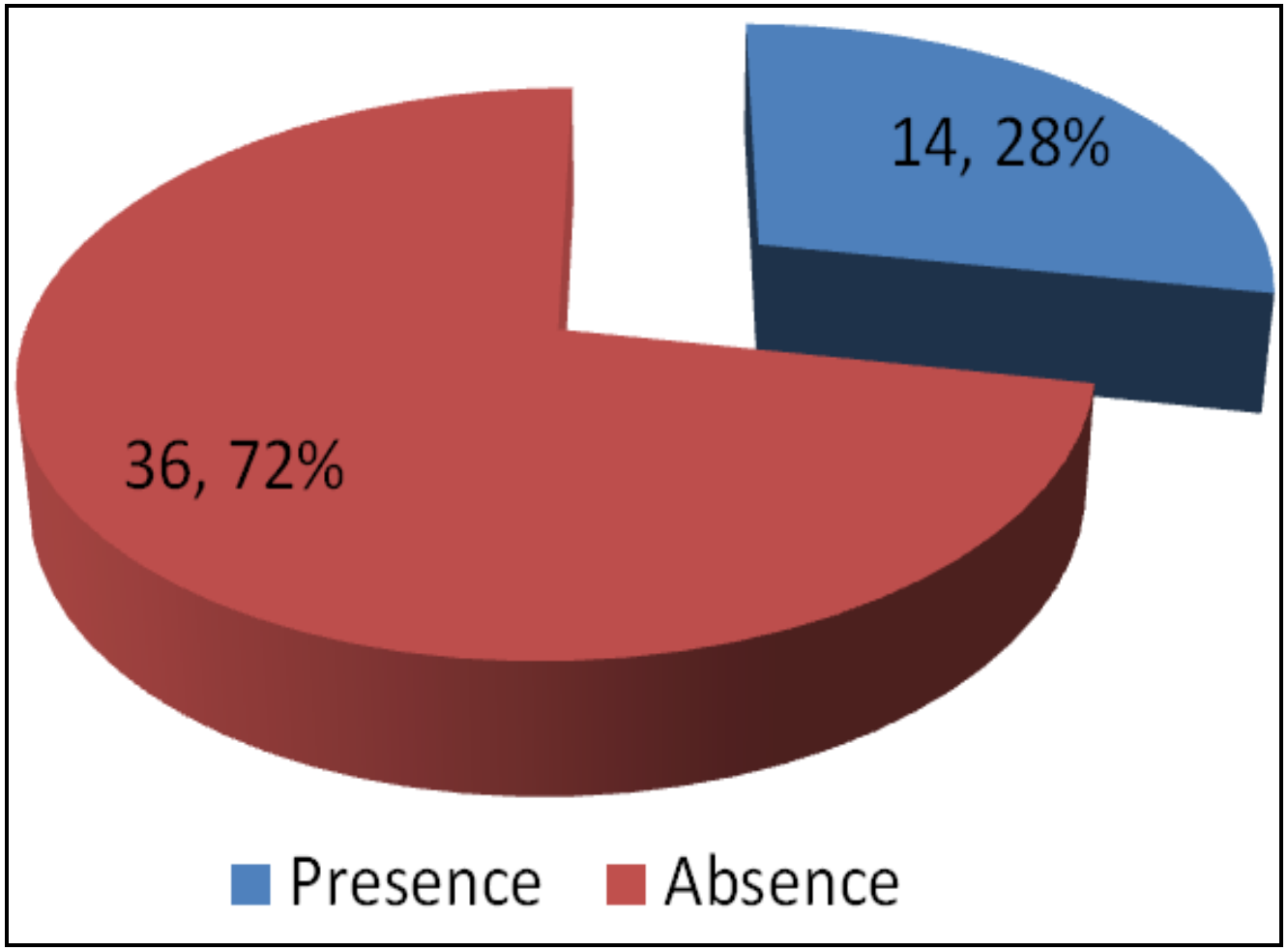


Descriptive Statistics

Parameter	n	%
CAD	50	
NO	36	72.0%
YES	14	28.0%

36 (72%) out of 50 patients were not a known CAD and 14 (28%) were known CAD patients and the diagrammatic representation as shown above.

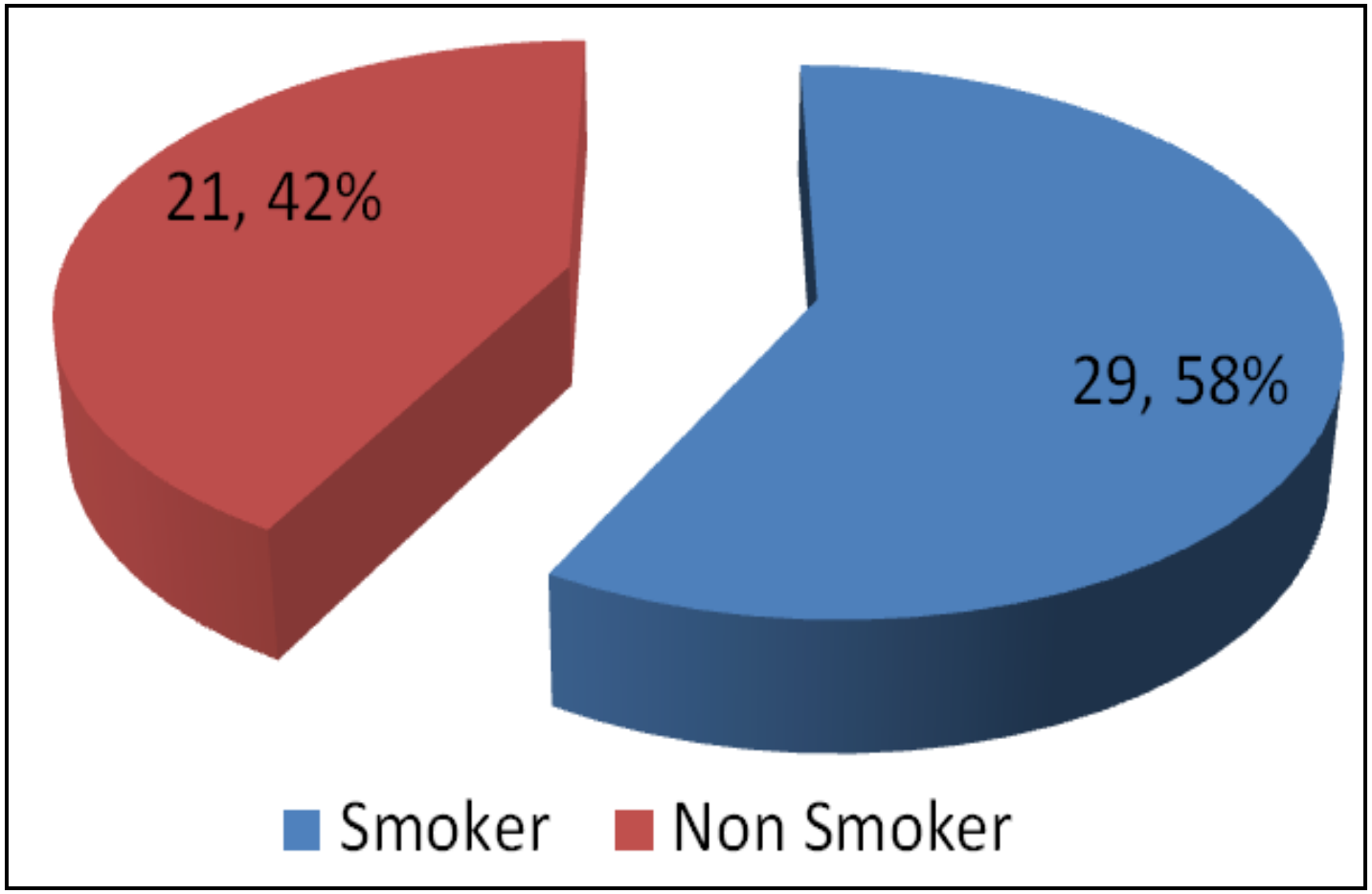
CAD



Descriptive Statistics

Parameter	n	%
Smoker	50	
NO	21	42.0%
YES	29	58.0%

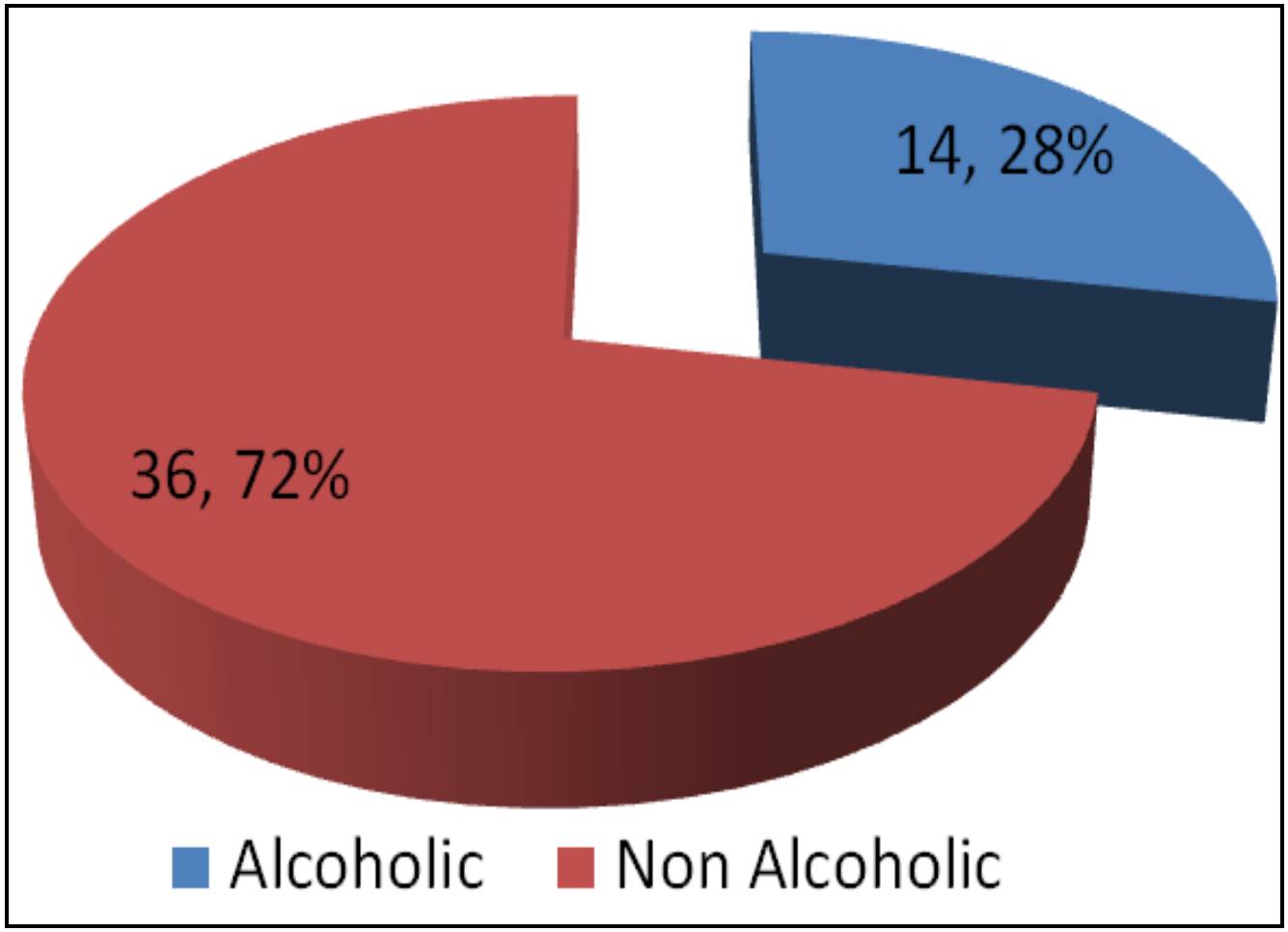
29 (58%) people out of 50 were smokers when compared to 21 (42%) people were nonsmokers.


21, 42%
29, 58%
Smoker
Non Smoker

Descriptive Statistics

Parameter	n	%
Alcoholic	50	
NO	36	72.0%
YES	14	28.0%

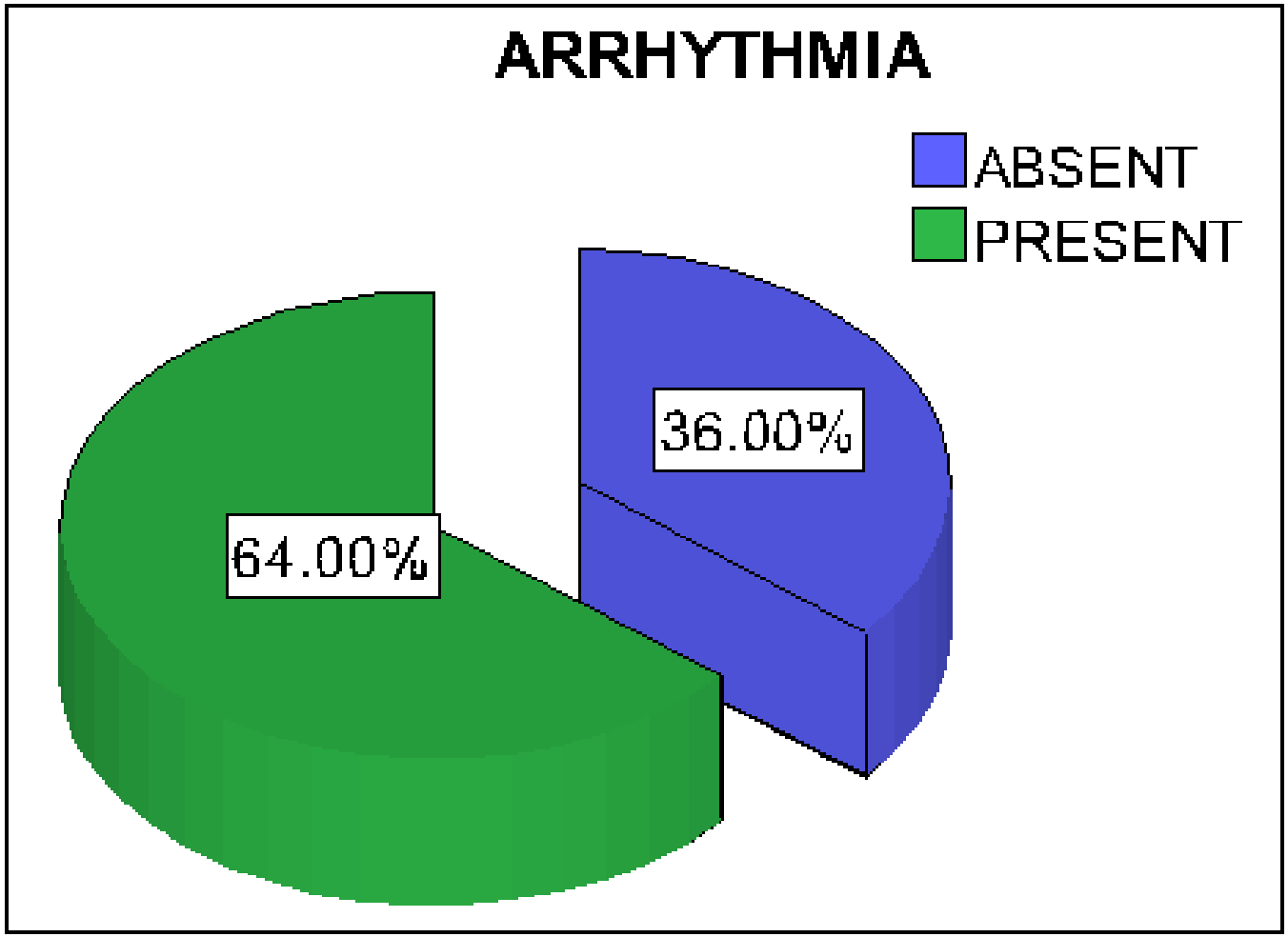
14 (28%) out of 50 patients were alcoholic and 36 (72%) were not a known alcoholic which is illustrated in the diagram above.


14, 28%
36, 72%
Alcoholic
Non Alcoholic

Descriptive Statistics

Parameter	n	%
ARRHYTHYMIA	50	
NO	18	36.0%
YES	32	64.0%

In this study 32 (64%) out of 50 MI patients developed arrhythmias during the stay in the hospital and 18 (36%) maintain normal sinus rhythm (had no arrhythmias) during the course of stay in the hospital.


ARRHYTHMIA
ABSENT
PRESENT
36.00%
64.00%

Descriptive Statistics

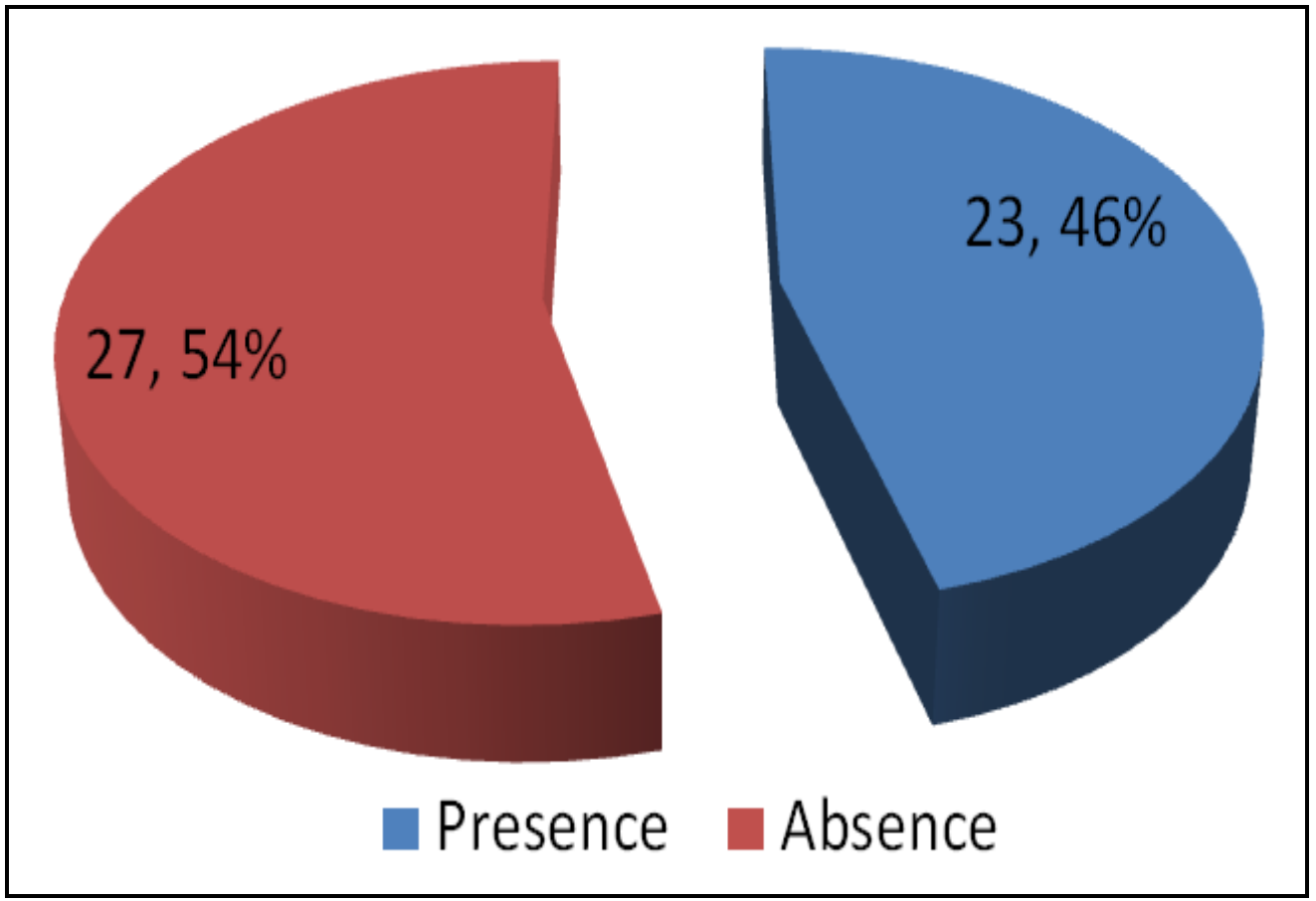
Parameter	n	%
DIAGNOSIS		
AWMI	50	
NO	27	54.0%
YES	23	46.0%
IWMI	50	
NO	28	56.0%
YES	22	44.0%
LWMI	50	
NO	47	94.0%
YES	3	6.0%
ANGI	50	
NO	48	96.0%
YES	2	4.0%

Multiple Responses on Diagnosis

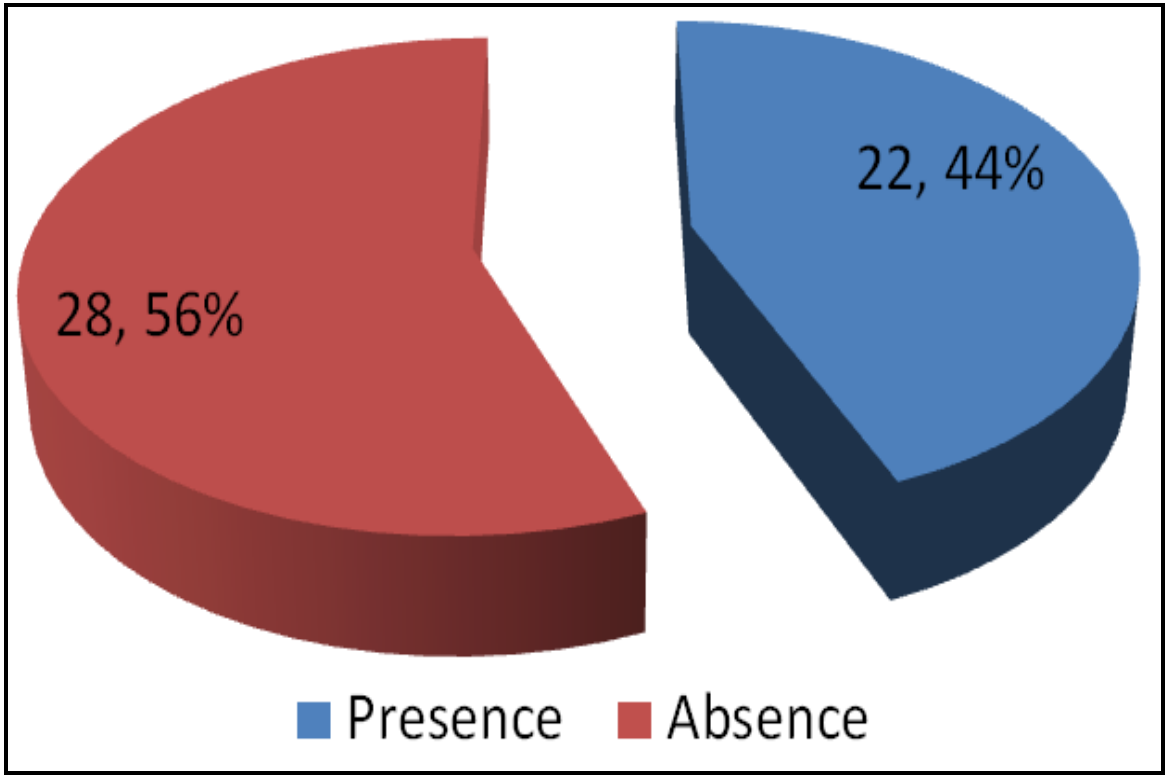
Diagnosis	n	%	% of Cases
AWMI	23	19.3%	46%
IWMI	22	18.5%	44%
LWMI	3	2.5%	6%
ANGI	2	1.7%	4%
Total	119	100.0%	238.0%

23 (46%) out of 50 patients had anterior wall MI.

AWMI

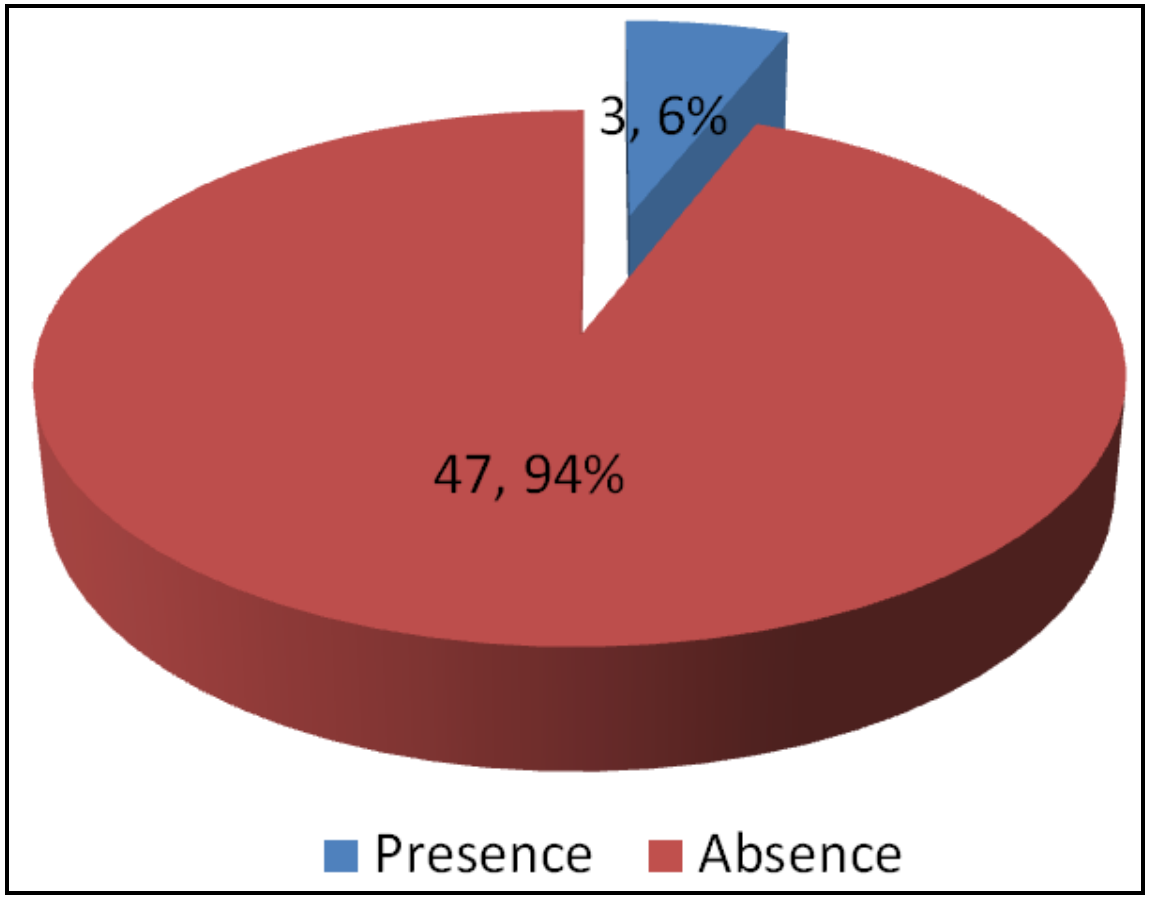


IWMI



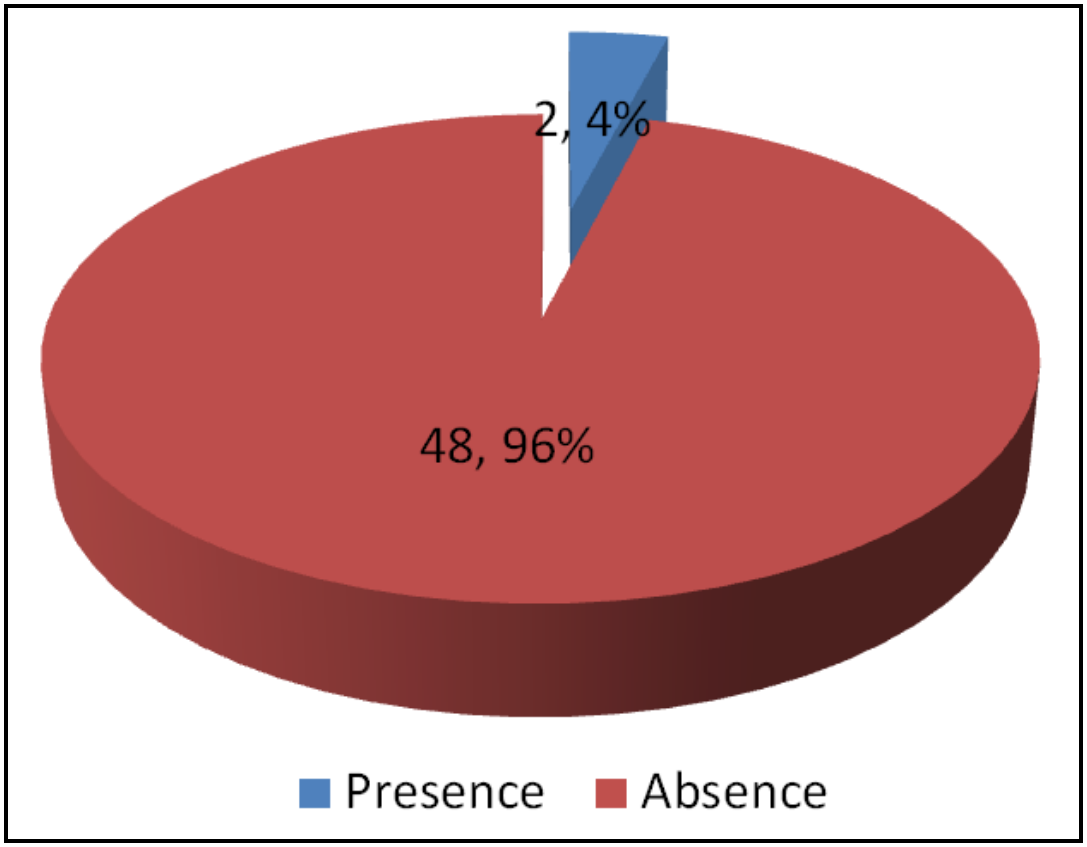
22 (44%) were inferior wall MI patients.

LWMI



3 (6%) patients had lateral wall MI.

UNSTABLE ANGINA



2 (4%) patients in the study suffered from unstable angina. The above diagrams illustrate the type of MI in the 50 patients under study.

Descriptive Statistics

Parameter	n	%
ARRHYTHYMIA TYPE	32	
1st Deg. Heart Block	3	9.4%
AF	5	15.6%
CHB	2	6.3%
LBBB	2	6.3%
PAC	2	6.3%
PBBB	2	6.3%
Sinus Brady Cardia	3	9.4%
SVT	3	9.4%
Torsedes de Pointes	1	3.1%
Ventricular Bigemini	1	3.1%
VPC	8	25.0%

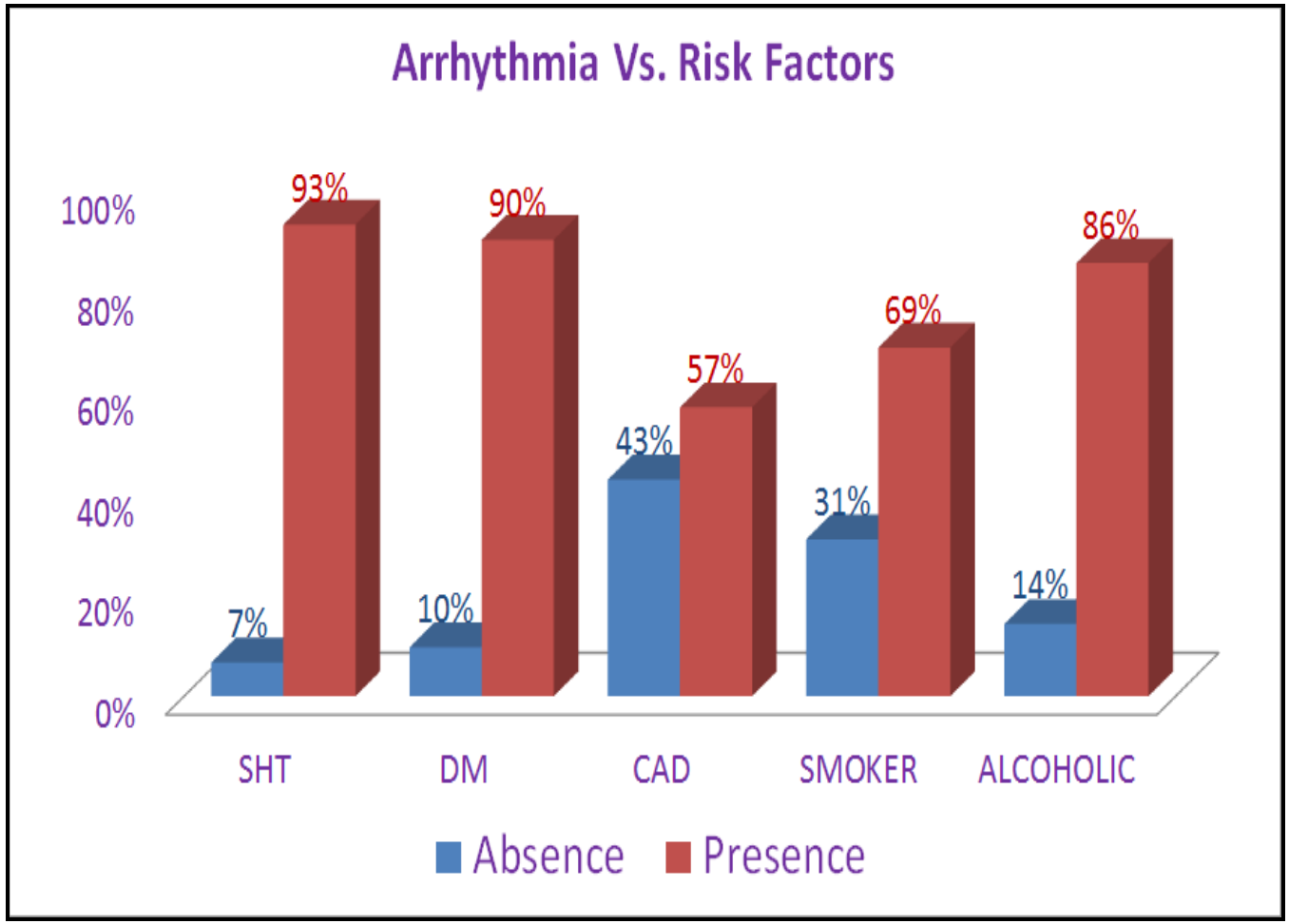
Of 32 patients who developed arrhythmias 3 (9.4%) had inferior wall MI, 5 patients (15.6%) developed atrial fibrillation, 2 (6.3%) developed complete heart block, 2 (6.3%) had LBBB, 2 (6.3%) had PAC, 2 (6.3%) had RBBB, 3 (9.4%) developed sinus bradycardia, 3

(9.4%) had SVT, 1 (3.1%) developed torsedes de pointes, 1 (3.1%) developed ventricular bigemini and 8 (25%) had VPC.

This is the distribution of type of arrhythmias in the study group. This shows that VPCs are most common among patients who develop MI.

Presence of Arrhythmia Vs. Risk Factors

		Arrhythmia		Total	Chi-square Statistics	p - Value
		No	Yes			
SHT	NO	16 (32%)	4 (8%)	20 (40%)	28.009	0.000
	YES	2 (4%)	28 (56%)	30 (60%)		
DM	NO	15 (30%)	4 (8%)	19 (38%)	24.533	0.000
	YES	3 (6%)	28 (56%)	31 (62%)		
CAD	NO	12 (24%)	24 (48%)	36 (72%)	0.397	0.529
	YES	6 (12%)	8 (16%)	14 (28%)		
SMOKER	NO	9 (18%)	12 (24%)	21 (42%)	0.739	0.390
	YES	9 (18%)	20 (40%)	29 (58%)		
Alcoholic	NO	16 (32%)	20 (40%)	36 (72%)	3.979	0.046
	YES	2 (4%)	12 (24%)	14 (28%)		
Total		18 (36%)	32 (64%)	50 (100%)		


Arrhythmia Vs. Risk Factors
100%
80%
60%
40%
20%
0%
93%
90%
57%
43%
69%
31%
86%
14%
7%
10%
SHT
DM
CAD
SMOKER
ALCOHOLIC
Absence
Presence

Of the 30 (60%) known systemic hypertensive patients 28 (56%) developed arrhythmias and 2 (4%) do not have arrhythmia. Using the Chi-square statistics T value is 0.000.

28 (56%) out of 31 (62%) diabetics had arrhythmias and 3 (6%) patients did not have arrhythmias and 4 (8%) out of 19 (38%) nondiabetics had arrhythmias. Here again using the Chi-square test P value is 0.000.

Of the known CAD patients 14 (28%), 8 (16%) had arrhythmias and among 36 (72%) non CAD patients 24 (48%) had arrhythmias.

Among 29 (58%) smokers, 20 (40%) had arrhythmias.

Out of 14 (28%) alcoholics, 12 (24%) had arrhythmias.

This tabular column and the diagrams illustrate that risk factors such as smoking, alcohol, hypertension, diabetes increases the risk of arrhythmias with P value being highly significant.

Presence of Arrhythmia Vs. Diagnosis

		Arrhythmia		Total	Chi-square Statistics	p - Value
		No	Yes			
Dia_AWMI	NO	9 (18%)	18 (36%)	27 (54%)	0.181	0.670
	YES	9 (18%)	14 (28%)	23 (46%)		
Dia_IWMI	NO	13 (26%)	15 (30%)	28 (56%)	3.004	0.083
	YES	5 (10%)	17 (34%)	22 (44%)		
Dia_LWMI	NO	16 (32%)	31 (62%)	47 (94%)	1.303	0.254
	YES	2 (4%)	1 (2%)	3 (6%)		
Dia_angi	NO	16 (32%)	32 (64%)	48 (96%)	3.704	0.054
	YES	2 (4%)	0 (0%)	2 (4%)		
Total		18 (36%)	32 (64%)	50 (100%)		

Presence of Arrhythmia Vs. Demographics

		Arrhythmia		Total	Chi-square Statistics	p - Value
		n (%)	n (%)			
		No	Yes			
GENDER	Female	2 (4%)	6 (12%)	8 (16%)	0.500	0.479
	Male	16 (32%)	26 (52%)	42 (84%)		
Age	Up to 30	1 (2%)	0 (0%)	1 (2%)	6.206	0.287
	31 - 40	3 (6%)	5 (10%)	8 (16%)		
	41 - 50	6 (12%)	4 (8%)	10 (20%)		
	51 - 60	5 (10%)	12 (24%)	17 (34%)		
	61 - 70	1 (2%)	6 (12%)	7 (14%)		
	> 70	2 (4%)	5 (10%)	7 (14%)		
Total		18 (36%)	32 (64%)	50 (100)		

There is not much significant difference in the incidence of arrhythmias among males and females and among different age groups.

There is no significant variation in the incidence of arrhythmias among the different types of MI.

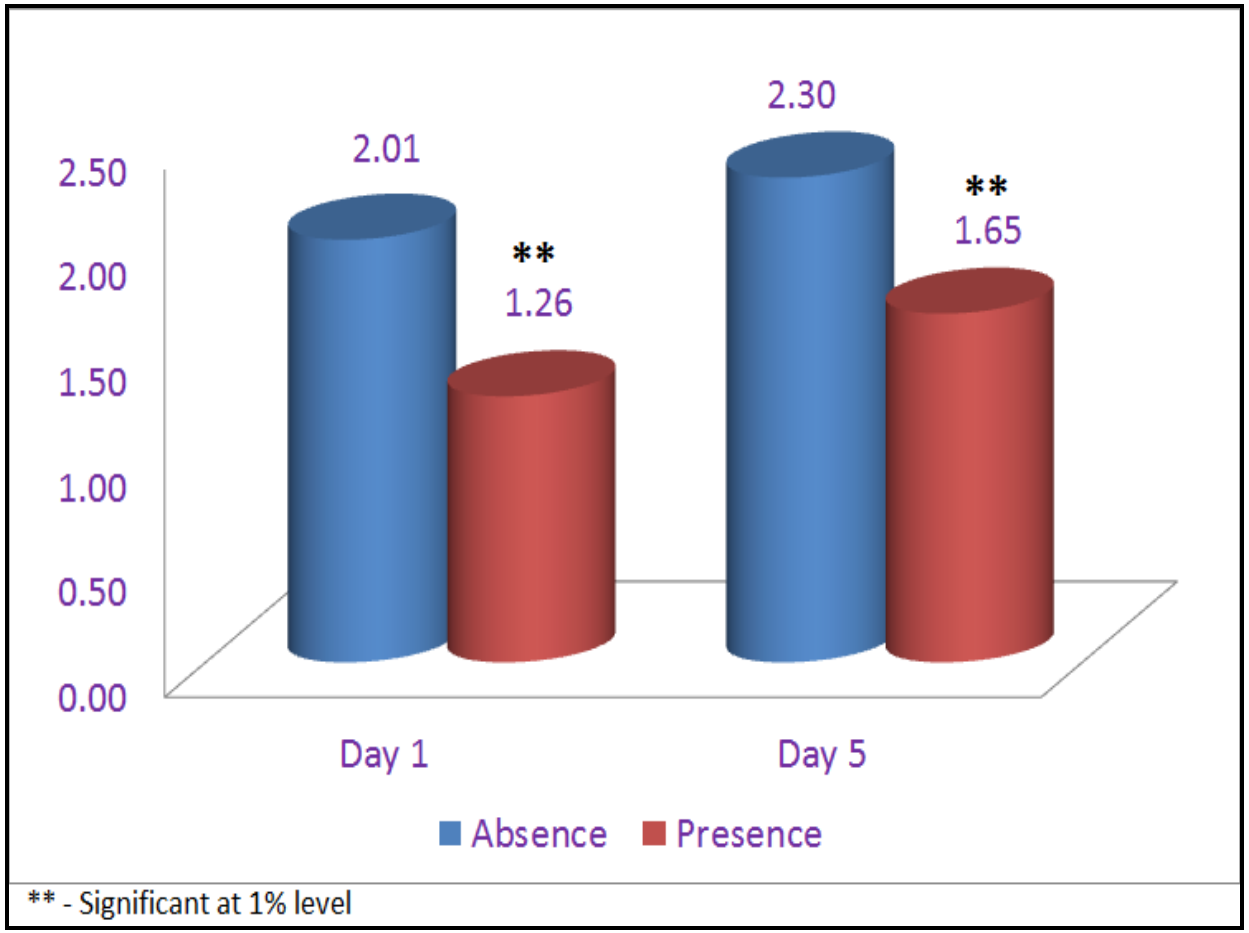
Paired Sample t Statistics

Pair	N	Mean	SD	Correlation	Mean Diff.	t - Statistics	P - Value
Day 1 Mg mEq/L	49	1.537	0.431	0.791**	-0.355	-8.567**	0.000
Vs.							
Day 5 Mg mEq/L	1	1.892	0.462				

** significant at 1% level
Incidence of arrhythmia in day 1 and day 5 is significantly high.

Out of 50 patients 32 of them had arrhythmias and using independent T statistics P value is highly significant. There is not much significant difference in the development of arrhythmia in relation to day following MI.

ARRHYTHMIA VS. MAGNESIUM DAY 1 AND DAY 5

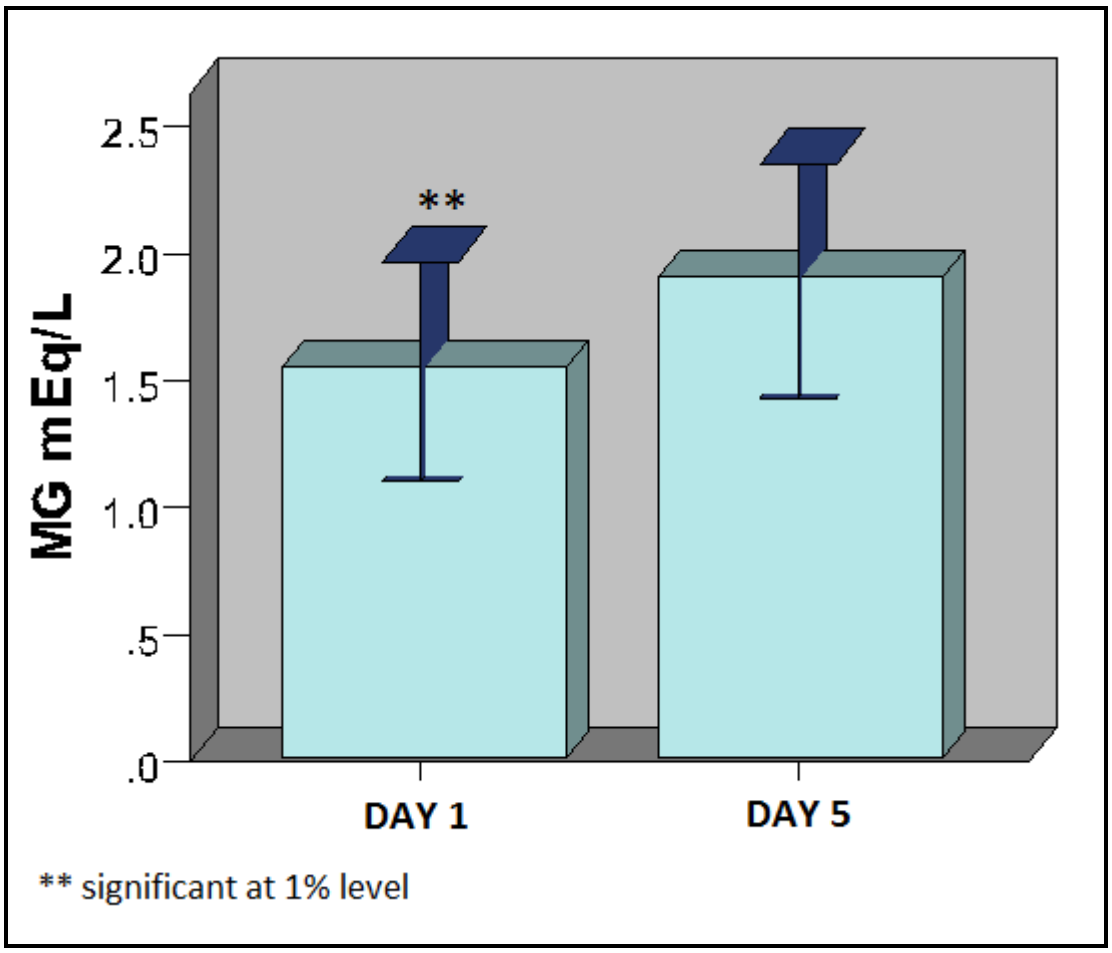


Independent t Statistics between presence and absence of Arrhythmia

Arrhythmia	N	Mean	SD	Mean Diff.	t Statistics	p Value
CHOLESTEROL						
No	18	194.9	27.7	0.639	0.080	0.936
Yes	32	194.3	26.6			
TGL						
No	18	145.7	28.4	4.979	0.677	0.502
Yes	32	140.7	22.9			
Day 1 Mg m Eq/L						
No	18	2.0	0.3	0.743	10.665**	0.000
Yes	32	1.3	0.2			
Day 5 Mg m Eq/L						
No	18	2.3	0.3	0.645	6.366**	0.000
Yes	32	1.7	0.4			

Arrhythmia	N	Mean	SD	Mean Diff.	t Statistics	p Value
Mg m Eq/L (Diff. from Day 5 to Day 1)						
No	18	0.3	0.143	-0.096	-1.118	0.269
Yes	31	0.4	0.345			
** significant at 1% level						

MAGNESIUM COMPARISON



Serum magnesium levels were low on the day of admission and progressively increased after that.

DISCUSSION

Magnesium has become a premier cardiovascular cation in the present decade.

Magnesium plays an important role in the pathogenesis of acute myocardial

Infarction as well as its complications. Magnesium is needed for activation of ATP which helps to maintain the sodium potassium pump and blocks calcium which has been implicated to the cause of arrhythmias in myocardial infarction patients.

In this study group of 50 patients, 42 were male and 8 were female. Of the 50 patients 30 of them were known hypertensives and 20 were nonhypertensives. The maximum incidence of myocardial infarction was between 40 and 50 years of age.

The serum magnesium level was calculated immediately on the day of admission for all the 50 patients and the mean serum magnesium level on day 1 was 1.3 for patients with arrhythmia and 2 for patients without arrhythmia.

Mean serum magnesium level on day 5 was 1.7 for patients who developed arrhythmia and 2.3 for patients who had no arrhythmia during hospital stay.

Dim truck[51] in his study of 67 patients with ischaemic heart disease showed a reduction in serum magnesium levels in the first three days of MI and the levels became normal by 15-25 days after MI.

In this present study serum magnesium levels of patients who developed arrhythmia are significantly low with a P value of 0.000. There was an increase in serum magnesium levels from day 1 to day 5 in both with and without arrhythmias.

Ventricular arrhythmias are more common than atrial arrhythmias.

CONCLUSION

This study was done in 50 patients who were being admitted to the cardiology department of Kilpauk Medical College during the period of April 2014 to September 2014. There were 42 males and 8 females included in the study. The most common risk factor found was hypertension, diabetes, smoking and alcohol.

The mean serum magnesium level on day 1 for patients with arrhythmia was 1.3 and 2 for patients without arrhythmia and the P value was highly significant with value of less than 0.000. On day 5 the mean serum magnesium levels with and without arrhythmias were 1.7 and 2.3 respectively again with a highly significant P value of 0.000.

The percentage of people who developed arrhythmia in the study was 64%. 60% were hypertensives and 62% were diabetics.

The distribution of type of MI were as follows

19.3% of them had AWMI, 18.5% had IWMI, 2.5% had LWMI and 1.7% had unstable angina. There was no significant difference in the incidence of arrhythmias among different types of MI.

Of the 60% of hypertensives 56% of them developed arrhythmias and among 62% of the diabetics 56% of them had

arrhythmias. The P value calculated by Chi-square test is 0.000. Of 28% of the known CADs 16% had arrhythmias and among 72% of the non CADs 48% had arrhythmias. There is no difference and the incidence of arrhythmias in CADs and non CADs.

With regard to smoking and alcohol there is significant difference in the incidence of arrhythmias.

Hence to conclude the incidence of arrhythmias in acute myocardial infarction is more in patients with low magnesium levels. The normal value being 1.5 mg/dl to 2.5 mg/dl. Diabetes, hypertension, smoking and alcohol are the major risk factors.

CLINICAL SIGNIFICANCE

Incidence of arrhythmia is about 90% in myocardial infarction patients and hence treating them with Magnesium Sulphate on the first day will prevent arrhythmias in the future. Oral Magnesium supplementation following Myocardial Infarction can be beneficial.

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PROFORMA

NAME: UNIT NO.:

AGE/SEX: I.P.NO.:

OCCUPATION: DATE OF ADMISSION:

ADDRESS: DATE OF DISCHARGE:

CONTACT NO:

COMPLAINTS:

Past history: Diabetes mellitus/systemic hypertension

GENERAL EXAMINATION

VITALS:

BP: PR: RR:

SYSTEM EXAMINATION:

INVESTIGATIONS:

Renal Profile

Random sugar	Urea	Creatinine

Sr. Sodium	Sr. Potassium

Sr. Magnesium within 24 hours of admission	Sr. Magnesium on Day of Discharge

Fasting Blood Sugar (In Diabetics)

Day 1	Day 2	Day 3	Day 4	Day 5

Fasting Lipid Profile

Total Cholesterol	Triglycerides

ECG In All Leads

DAY 1	DAY 2	DAY 3	DAY 4	DAY 5

2D ECHOCARDIOGRAPHY:

Signature of Investigator **Signature of Guide**

ABBREVIATIONS USED

CCF	Congestive cardiac failure
AMI	Acute myocardial infarction
CAD	Coronary artery disease
IHD	Ischaemic heart disease
CABG	Coronary artery bypass grafting
LBBB	Left bundle branch block
PCI	Percutaneous coronary intervention
NSTEMI	Non ST elevation Myocardial Infarction
AWMI	Anterior wall myocardial infarction
IWMI	Inferior wall myocardial infarction
LWMI	Lateral wall myocardial infarction
LVH	Left ventricular hypertrophy
HOCM	hypertrophic obstructive cardiomyopathy
DM	Diabetes mellitus
CPR	cardio pulmonary rescucitation
AF	Atrial fibrillation
LAFB	Left anterior fascicular block
SVT	Supra ventricular tachycardia
LAD	Left anterior descending

Name	Age	Sex	IP Number	DOA	DOD	SHT	DM	CAD	Smoker	Alcoholic	Cholesterol	TGL	Day of arrhythm
Mr. L. Mansoor	55	Male	1409053	1/4/14	5/4/14	No	Yes	No	Yes	No	236	172	No arrhythmia
Mr. Ammavasai	65	Male	1408814	2/4/14	6/4/14	Yes	Yes	No	Yes	Yes	240	152	3
Mr. Dass	46	Male	1409150	2/4/14	6/4/14	Yes	No	Yes	No	No	200	156	1
Mr. Subramani	73	Male	1409319	3/4/14	7/4/14	Yes	Yes	No	No	No	222	148	1
Mr. Sundaram	73	Male	1409568	4/4/14	9/4/14	No	No	No	Yes	No	206	152	No arrhythmia
Mr. Ravikumar	65	Male	1409553	5/4/14	10/04/14	Yes	No	No	Yes	No	220	158	No arrhythmia
Mr. Thillai Ganesh	51	Male	1409611	5/4/14	10/4/14	Yes	Yes	Yes	Yes	No	238	162	3
Mr. Kumar	48	Male	1409658	6/4/14	10/4/14	No	No	No	Yes	No	200	148	No arrhythmia
Mrs. Geetha	38	Female	1409622	6/4/14	10/4/14	Yes	Yes	No	No	No	186	150	2
Mr. Kantha Rao	44	Male	1409736	7/4/14	11/4/14	Yes	Yes	No	Yes	No	210	154	2
Mr. Selvam	46	Male	1410066	9/4/14	14/4/14	No	No	No	No	No	172	150	No arrhythmia
Mr. Munusamy	60	Male	1410274	11/4/14	15/4/14	Yes	Yes	Yes	Yes	No	200	154	1
Mr. Kaliappan	40	Male	1410319	11/4/14	16/4/14	Yes	Yes	Yes	Yes	No	216	160	1
Mr. Manimaran	25	Male	1410739	15/4/14	19/4/14	No	No	No	No	No	182	149	No arrhythmia
Mr. Ramamurthy	59	Male	1410974	18/4/14	23/4/14	Yes	Yes	No	Yes	Yes	204	154	1
Mrs. Chokammal	75	Female	1411328	21/4/14	25/4/14	Yes	Yes	No	No	No	216	150	1
Mr. Murugan	75	Male	1411480	23/4/14	27/4/14	No	No	Yes	Yes	No	182	146	No arrhythmia
Mrs. Meenakshi	60	Female	1411223	21/4/14	26/4/14	Yes	No	No	No	No	202	158	2
Mrs. Kumari	60	Female	1411660	24/4/14	29/4/14	No	Yes	Yes	No	No	178	152	No arrhythmia
Mr. Velayutham	63	Male	1411763	25/4/14	29/4/14	No	Yes	No	Yes	No	190	138	2
Mr. Rajasekar	40	Male	1411427	27/4/14	1/5/14	Yes	Yes	No	Yes	Yes	175	150	1
Mr. Parthasarthy	57	Male	14123114	30/4/14	1/5/14	Yes	Yes	Yes	Yes	No	208	179	1
Mr. Sowrimuthu	56	Male	14112464	1/5/14	4/5/14	No	No	Yes	No	No	222	180	No arrhythmia
Mr. Kareem	54	Male	1412881	5/5/14	9/5/14	Yes	Yes	No	Yes	No	181	139	1
Mr. Krishnamurthy	56	Male	1412580	2/5/14	7/5/14	Yes	Yes	No	Yes	Yes	214	156	1
Mr. Selvaraj	65	Male	141276	4/5/14	7/5/14	Yes	No	Yes	Yes	No	190	138	4
Mr. Ravindran	54	Male	1413058	7/5/14	12/5/14	No	No	Yes	Yes	Yes	208	154	No arrhythmia
Mrs. Malathy	40	Female	1413783	8/5/14	13/5/14	Yes	Yes	No	No	No	209	157	2
Mrs. Gunasundari	40	Female	1413488	8/5/14	14/5/14	Yes	Yes	No	No	No	199	155	No arrhythmia
Mr.Ponnambala	47	Male	1413220	8/5/14	12/05/14	No	Yes	No	No	No	200	138	3
Mr. Suman	46	Male	1413567	10/5/14	14/5/14	Yes	Yes	No	Yes	Yes	209	162	2
Mr. Vembu	65	Male	1413104	7/5/14	13/5/14	Yes	Yes	No	Yes	Yes	168	132	1
Mr. Irudayaraj	38	Male	1414252	16/05/14	21/05/14	No	No	Yes	Yes	No	201	199	No arrhythmia
Mr. Shanmugam	36	Male	1413980	13/05/14	19/05/14	Yes	No	Yes	No	No	208	150	3

Name	Age	Sex	IP Number	DOA	DOD	SHT	DM	CAD	Smoker	Alcoholic	Cholesterol	TGL	Day of arrhythm
Mr. Murali	50	Male	1414414	17/05/14	22/05/14	No	No	No	No	No	178	100	No arrhythmia
Mr. Govindan	75	Male	1414307	17/05/14	24/05/14	Yes	Yes	No	Yes	Yes	128	88	3
Mr. Prakash	35	Male	1414962	22/05/14	28/05/14	No	No	No	Yes	No	172	110	No arrhythmia
Mr. Chinnaraj	56	Male	1414960	22/05/14	26/05/14	Yes	Yes	No	Yes	Yes	138	78	1
Mr. Srinivasan	54	Male	1415272	25/05/14	29/05/14	Yes	Yes	No	No	No	205	100	1
Mr. Hasi Bul	54	Male	1415477	26/05/14	29/05/14	No	Yes	No	Yes	Yes	190	132	2
Mr. Balmurugan	43	Male	1415880	28/05/14	31/05/14	No	No	No	No	No	168	115	No arrhythmia
Mr. Vadivel	65	M ale	1416100	29/05/14	4/6/14	Yes	Yes	No	Yes	No	138	133	1
Mr. Jony Basha	50	Male	1417519	13/06/14	18/06/14	No	No	No	No	No	152	90	No arrhythmia
Mr. Anandhan	51	Male	1418575	21/06/14	24/06/14	No	Yes	No	No	No	187	130	2
Mrs. Saraswathy	57	Female	1419007	25/06/14	30/06/14	Yes	Yes	No	No	Yes	221	159	3
Mr. Pichai	75	Male	1419118	3/7/14	7/7/14	Yes	Yes	Yes	Yes	Yes	165	99	1
Mr. Selvaraj	62	Male	1419120	4/7/14	9/7/14	Yes	Yes	No	Yes	Yes	191	121	1
Mrs. Pattamal	80	Female	1419225	5/7/14	10/07/14	Yes	Yes	No	No	No	167	125	2
Mr. Joseph	42	Male	1419230	8/7/14	13/07/14	No	No	No	No	No	170	122	No arrhythmia
Mr. Balaji	55	Male	1419233	11/7/13	16/07/14	No	No	Yes	Yes	Yes	262	170	No arrhythmia

Diagnosis	Mg mEq/L		Echo	Arrhythymia	Type of arrhytyhmia
	Day 1	Day 5			
DM type 2 SHT/EXT. AWMI	2	2.5	RWMA+, EF 48%, Mod LV dysfunction	No	-
DM/SHT/IWMI	1.4	1.8	No RWMA, normal LV function, EF 60%	Yes	VPC
CAD/IWMI	1	1.5	RWMA+, EF 38%	Yes	1st degree HB
DM/SHT/AWMI	1.2	1.8	No RWMA, normal LV function	Yes	S. Bradycardia
DM/IWMI	2	2.5	RWMA+, EF 40%	No	-
DM/SHT/AWMI/	2.5	2.7	Mild LVH+, mild hypokinesia+, EF 48%	No	-
SHT/CAD/IWMI	1.4	1.6	LVH+, EF 52%	Yes	VPC
AWMI	2	2.2	No RWMA, normal LV sys function	No	-
DM/IWMI	1.2	1.7	No RWMA, normal LV sys function	Yes	RBBB
DM/SHT/Ext. AWMI	1.3	1.9	RWMA+, EF 40%	Yes	VPC
DM/SHT/Ext. AWMI	2	2.3	LVH+, No RWMA, EF 70%	No	-
CAD/Ext./ AWMI	1.1	1.5	Tachycardia during study, No RWMA	Yes	AF
SHT/CAD/IWMI	1.2	1.8	Severe RWMA+ EF 30%, Severe LV	Yes	1st degree heart block
AWMI	2.4	2.5	No RWMA, normal systolic function	No	-
DM/SHT/IWMI	1	1.5	RA/RV dilated, EF 50%	Yes	SVT
DM/SHT/AWMI	1.4	1.8	No RWMA, normal LV function	Yes	AF
DM/SHT/CAD/LWMI	2.4	2.7	No RWMA, normal LV function	No	-
SHT/IWMI	1.5	1.9	No RWMA, normal LV function	Yes	Ventricular bigemini
DM/SHT/CAD/AWMI	1.8	2.3	No RWMA, normal LV function	No	-
DM/IWMI	1.1	1.5	RWMA+/EF 48%/mild LV dysfunction	Yes	1st degree heart block
Ac. IWMI	1.4	2	No RWMA, normal LV function	Yes	PAC
SHT/CAD/IWMI	1.2	1.6	RWMA+, EF 37%,	Yes	LBBB
CAD/Ac. IWMI	1.8	2.2	No RWMA, normal LV function	No	-
DM/IWMI	1	1.5	RWMA+, EF 50%, mild LV dysfunction	Yes	AF
DM/SHT/IWMI	1.3	1.8	No RWMA, normal LV function	Yes	VPC
IWMI	1.1	-	No RWMA	Yes	CHB
DM/SHT/CAD/LWMI	2	2.2	No RWMA, LVH	No	-
DM/AWMI	1.3	2	No RWMA, normal LV function	Yes	RBBB
SHT/DM/AWMI	2.2	2.5	NoRWMA, LVH+, normal LV function	No	-
DM/AWMI	1.7	1.3	RWMA+, moderate LV dysfunction	Yes	VPC
SHT/DM/AWMI	1	1.6	No RWMA, normal LV function	Yes	SVT
DM/AWMI	1.3	2	No RWMA, normal LV function	Yes	LBBB
CAD/ASMI	2.3	2.4	No RWMA, normal LV function	No	-
SHT/IWMI	1.3	1.6	RWMA+, moderate LV dysfunction	Yes	Sinus Bradycardia

Diagnosis	Mg mEq/L		Echo	Arrhythymia	Type of arrhytyhmia
	Day 1	Day 5			
IWMI	1.5	1.7	RWMA+, mild MR, mild LV	No	-
SHT/DM, IWMI	1.2		No RWMA, normal LV function	Yes	CHB
SHT/DM, Unstable angina	2	2.3	No RWMA, normal LV function	No	-
AWMI	1	1.3	RWMA+, hypokinesia of	Yes	Torsedes de pointes
DM/IWMI	1.1	1.5	RWMA+, severe LV dysfunction	Yes	AF
AWMI	1.4	2	No RWMA, normal LV function	Yes	VPC
SHT/DM/evolved IWMI	1.6	2	No RWMA, normal LV function	No	-
SHT/AWMI	1.1	1.4	RWMA+, severe LV dysfunction	Yes	SVT
AWMI	1.8	2	No RWMA, normal LV function	No	-
DM/IWMI	1.3	2	Hypokinesia +, RWMA+	Yes	Sinus bradycardia
SHT/DM/Ac. AWMI	1.2	1.7	No RWMA, normal LV function	Yes	PAC
SHT/DM/LWMI	1.4	1.9	No RWMA, normal LV function	Yes	VPC
SHT/DM/AWMI	2	2.2	No RWMA, normal LV function	Yes	AF
SHT/DM/AWMI	1.3	1.6	RWMA+, mild LV dysfunction	Yes	VPC
unstable angina	2	2.1	No RWMA, normal LV function	No	-
DM/SHT/IWMI	1.8	2.3	RWMA+, severe LV dysfunction	No	-

INSTITUTIONAL ETHICAL COMMITTEE
GOVT.KILPAUK MEDICAL COLLEGE,
CHENNAI-10
Ref.No.2212/ME-1/Ethics/2014 Dt:03.04.2014.
CERTIFICATE OF APPROVAL

The Institutional Ethical Committee of Govt. Kilpauk Medical College, Chennai reviewed and discussed the application for approval "A Study on Serum magnesium levels in acute myocardial infarction in relation to arrhythmias in patients admitted in Govt, Kilpauk Medical College & Hospital" - For Project work submitted by Dr.Prabha.G, MD (GM), PG Student, KMC, Chennai-10.

The Proposal is APPROVED.

The Institutional Ethical Committee expects to be informed about the progress of the study any Adverse Drug Reaction Occurring in the Course of the study any change in the protocol and patient information /informed consent and asks to be provided a copy of the final report.

GOVT. KILPAUK MEDICAL COLLEGE
30 MAY 2014
Kilpauk, Chennai-10.

30/5/14.
CHAIRMAN,
Ethical Committee
Govt.Kilpauk Medical College,Chennai